Acute Hypoxemic Respiratory Failure

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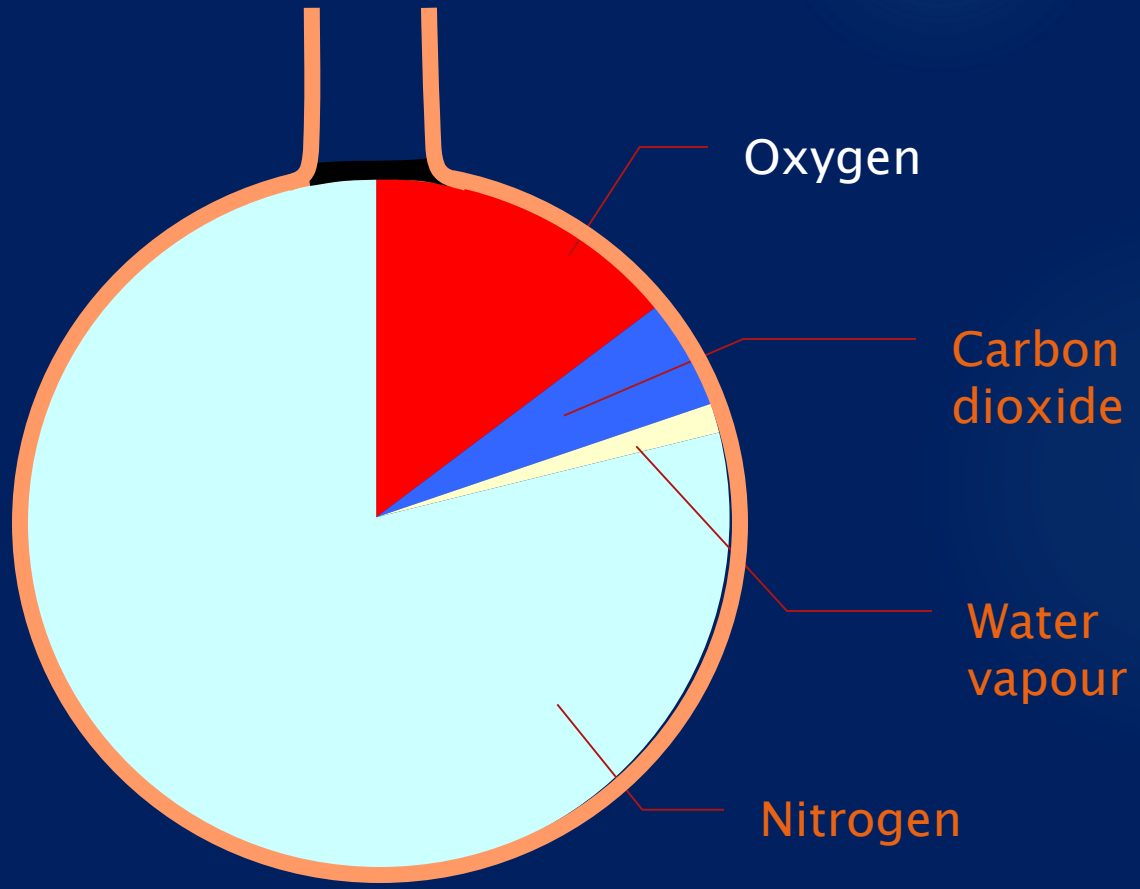
PULMONARY AND CRITICAL CARE MEDICINE

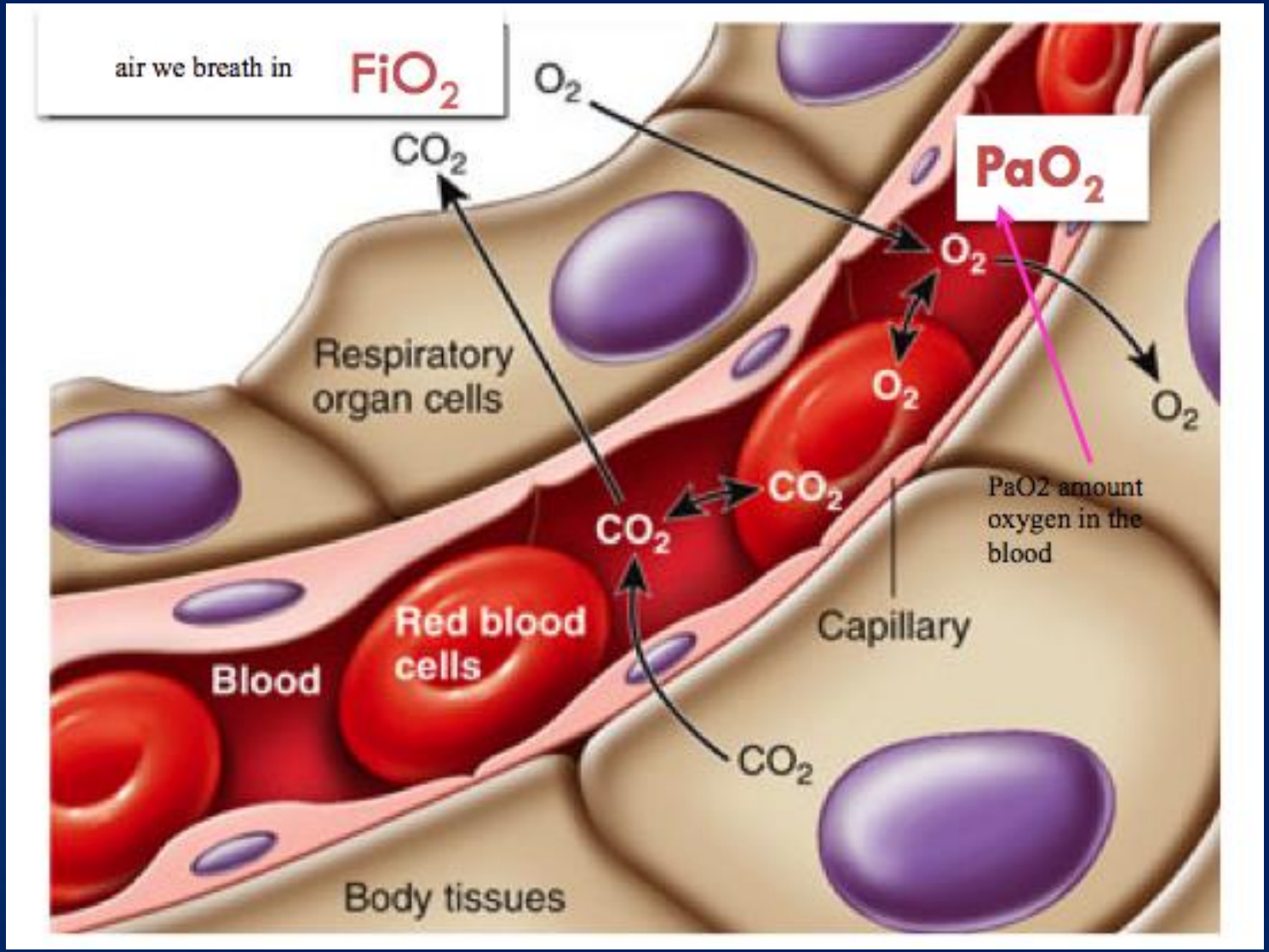
- Hypoxemic Respiratory Failure
- Arterial Po2 of < 60 mm Hg

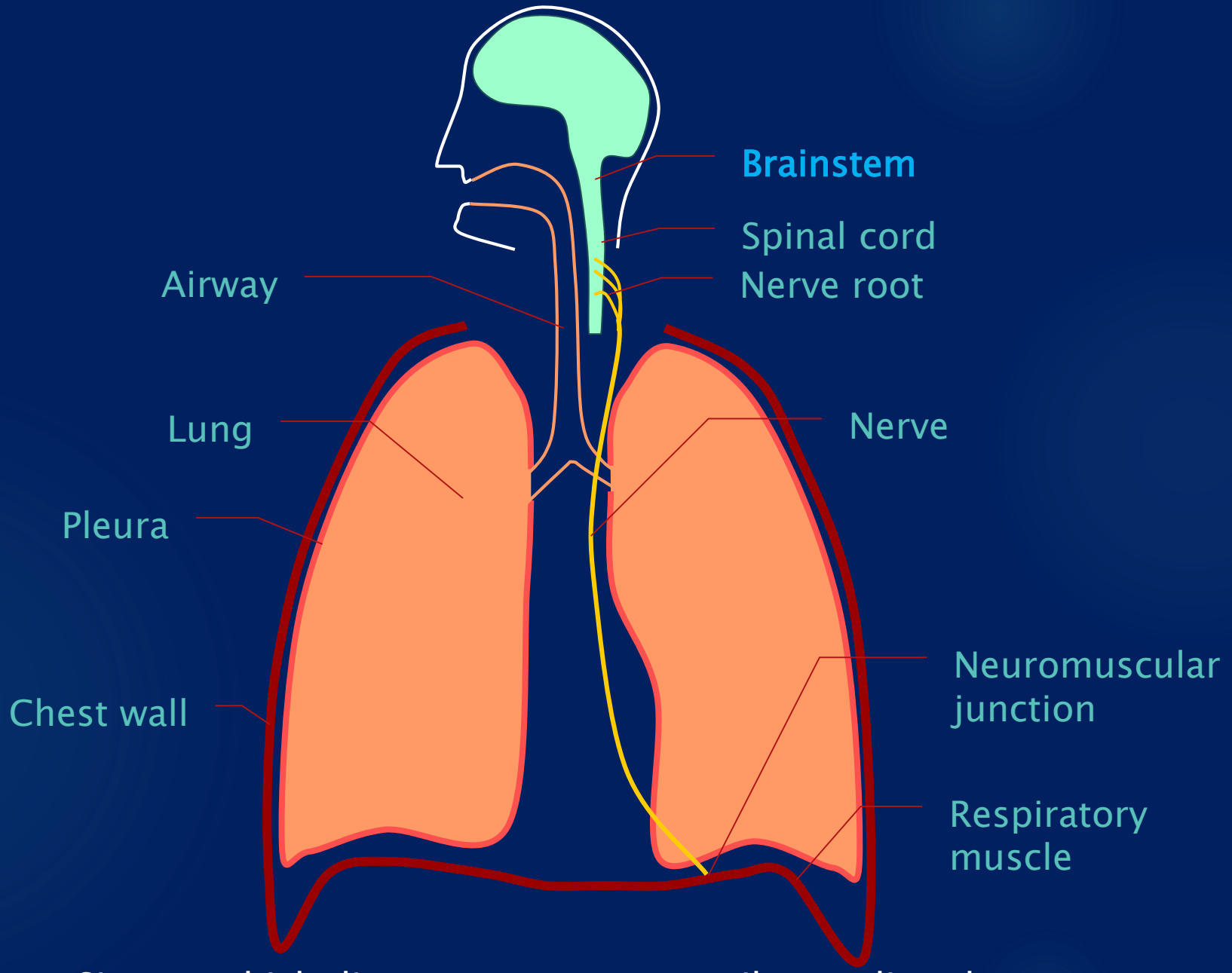
- Hypercapnic Respiratory Failure
- Pco2 > 45 mm Hg



$$\text{Alveolar pressure} = PAO2 + PACO2 + PAH2O + PAN2$$

Fio2 & Pao2


air we breath in
FiO_2
O_2
CO_2
PaO_2
O_2
Respiratory organ cells
O_2
O_2
CO_2
CO_2
PaO2 amount oxygen in the blood
Red blood cells
Blood
Capillary
CO_2
Body tissues



Sites at which disease may cause ventilatory disturbance

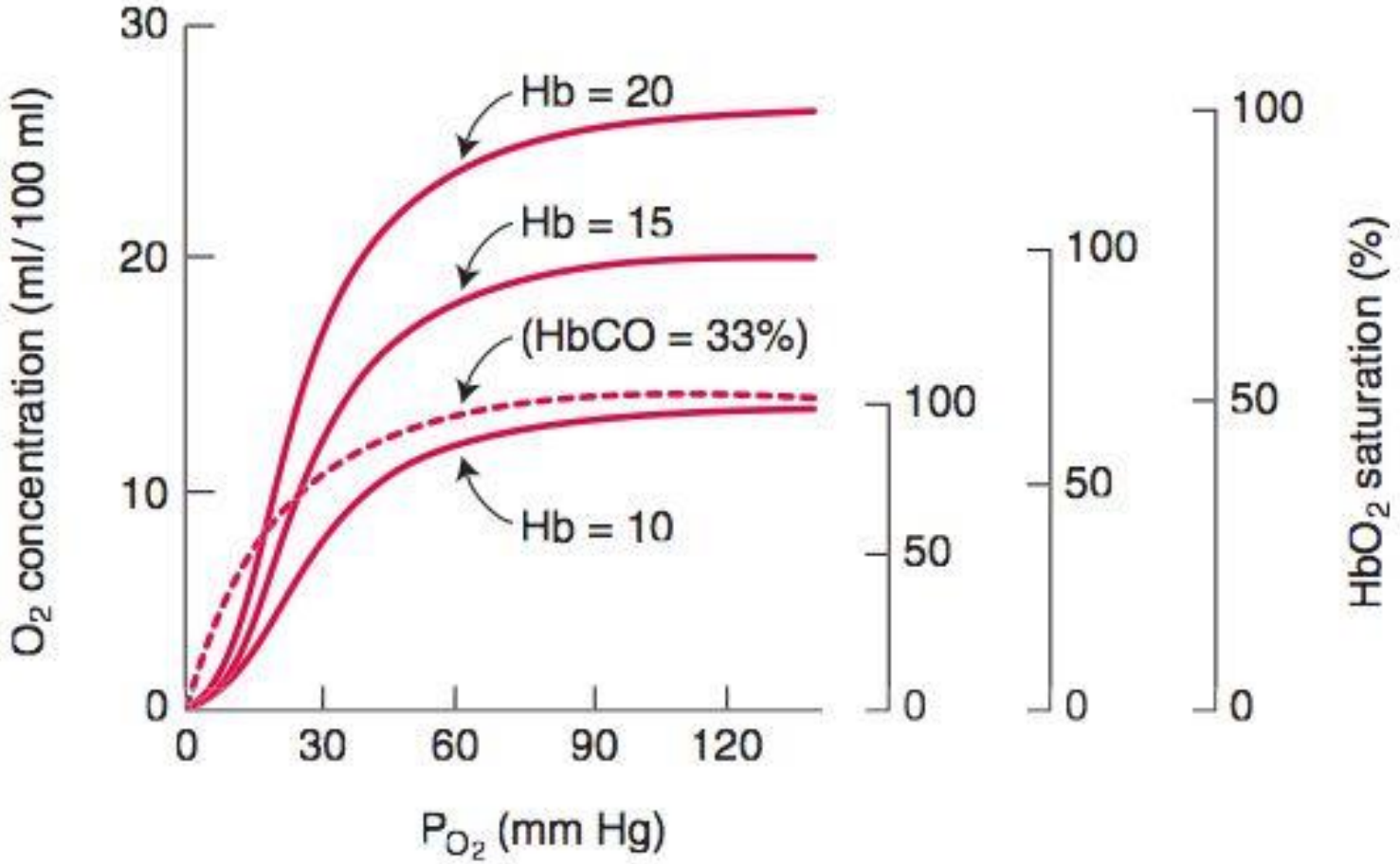


Figure 6-2. Effects of anemia and polycythemia on O_2 concentration and saturation. In addition, the *broken line* shows the O_2 dissociation curve when one-third of the normal hemoglobin is bound to CO. Note that the curve is then shifted to the left.

Oxygen partial pressure (mm Hg)

Hypoxemic respiratory failure

- Acute (over hours to days)
- Chronic (over weeks to months)

- ***Oxygen delivery to the tissues***

- **cardiac output and oxygen content**

Oxygen delivery (DO_2)

- rate at which oxygen is transported from the lungs to the microcirculation:

- DO_2 (mL/min) = Q x CaO_2
- Q is the cardiac output
- Normal DO_2 is approximately 1000 mL/min

CaO_2 (mL O_2 /dL) = (1.34 x hemoglobin concentration x SaO_2) + (0.0031 x PaO_2)

Normal CaO_2 is approximately 20 mL O_2 /dL

Hypoxemic respiratory failure

- Anemic patient
- patients with very low cardiac output

- Tissue hypoxia may exist despite a seemingly adequate arterial Po2

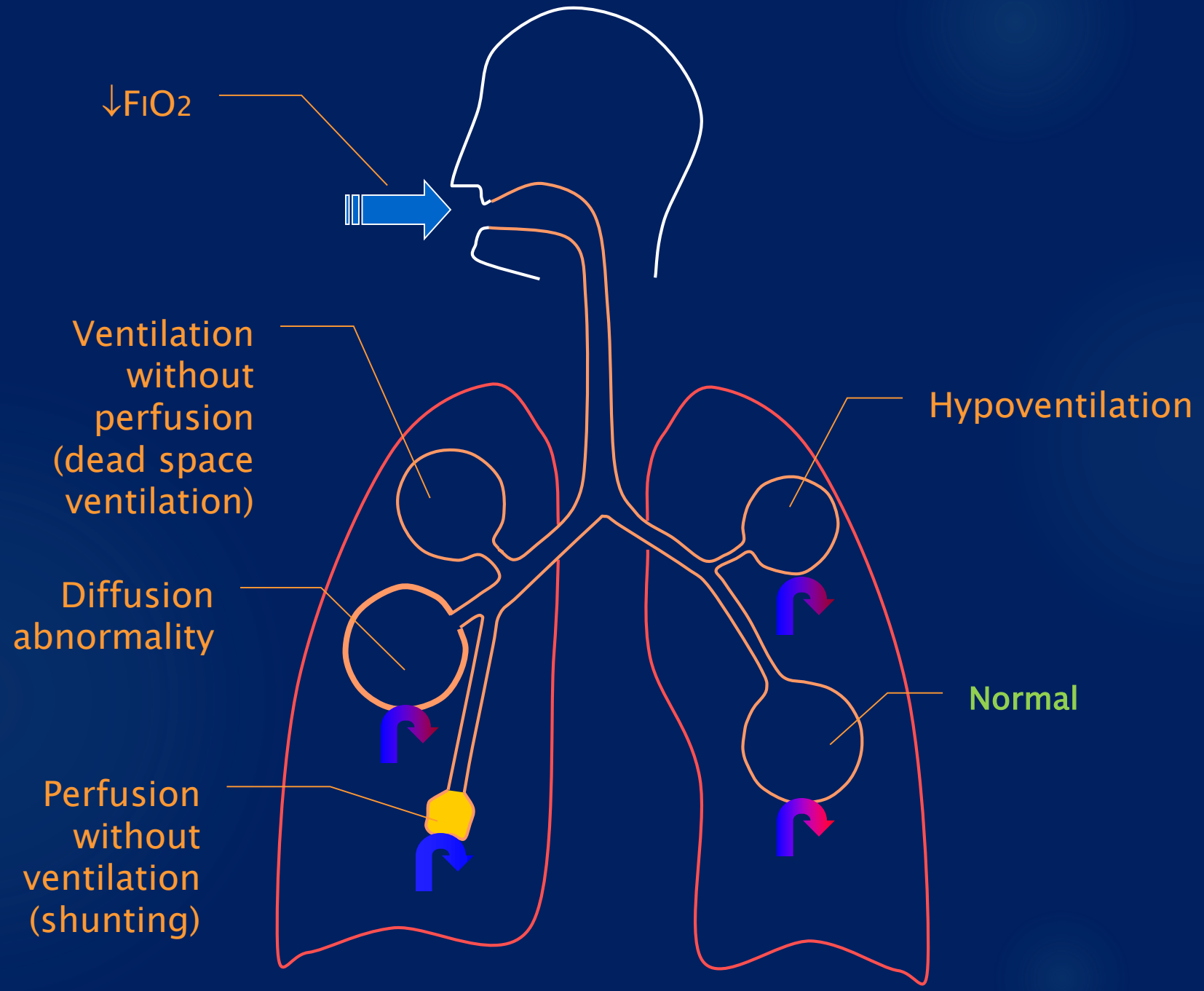
Hypoxemic respiratory failure

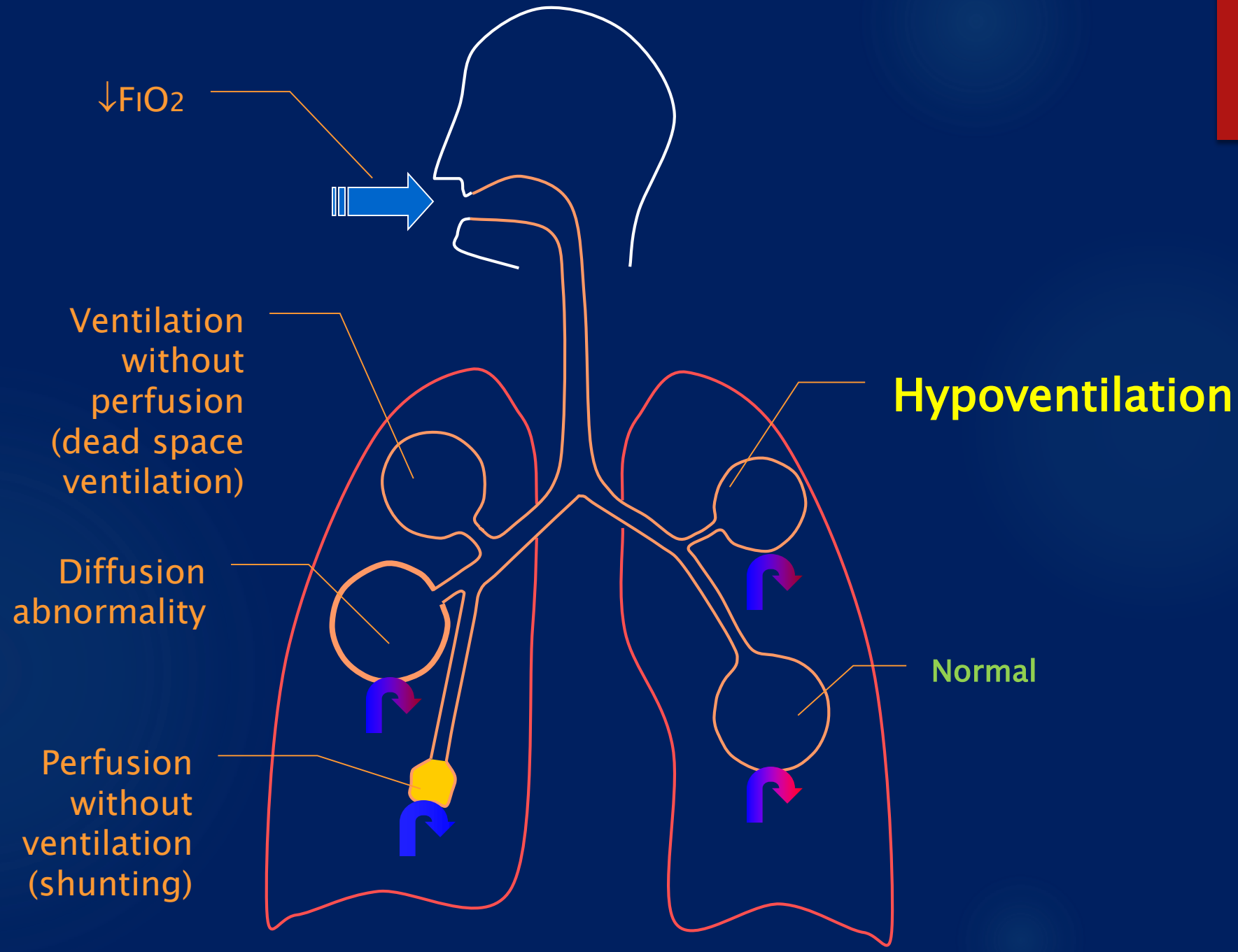
- both hypoxemic and hypercarbic respiratory failure may coexist

Hypoventilation

MECHANISMS OF HYPOXEMIA

1. ↓ inspired Po2
2. Hypoventilation
3. impaired diffusion
4. ventilation-perfusion (V/Q) mismatch
5. right-to-left shunt


↓FIO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal


↓FIO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal

Hypoventilation

- ↑ PaCO2 & PACO2
- ↓ PAO2

As a result

- ↓ diffusion of oxygen from the alveolus to the pulmonary capillary

Net effect is hypoxemia

Hypoventilation

- **A-a gradient is usually normal**
- **A-a oxygen gradient = PAO2 - PaO2**

- **PaO2 is measured by ABG**

- PAO2 is calculated using the alveolar gas equation:
- PAO2 = (FiO2 x [Patm - PH2O]) - (PaCO2 ÷ R)

- respiratory quotient = 0.8 at steady state
- relative utilization of carbohydrate, protein, and fat

A-a gradient = 2.5 + 0.21 x age in years

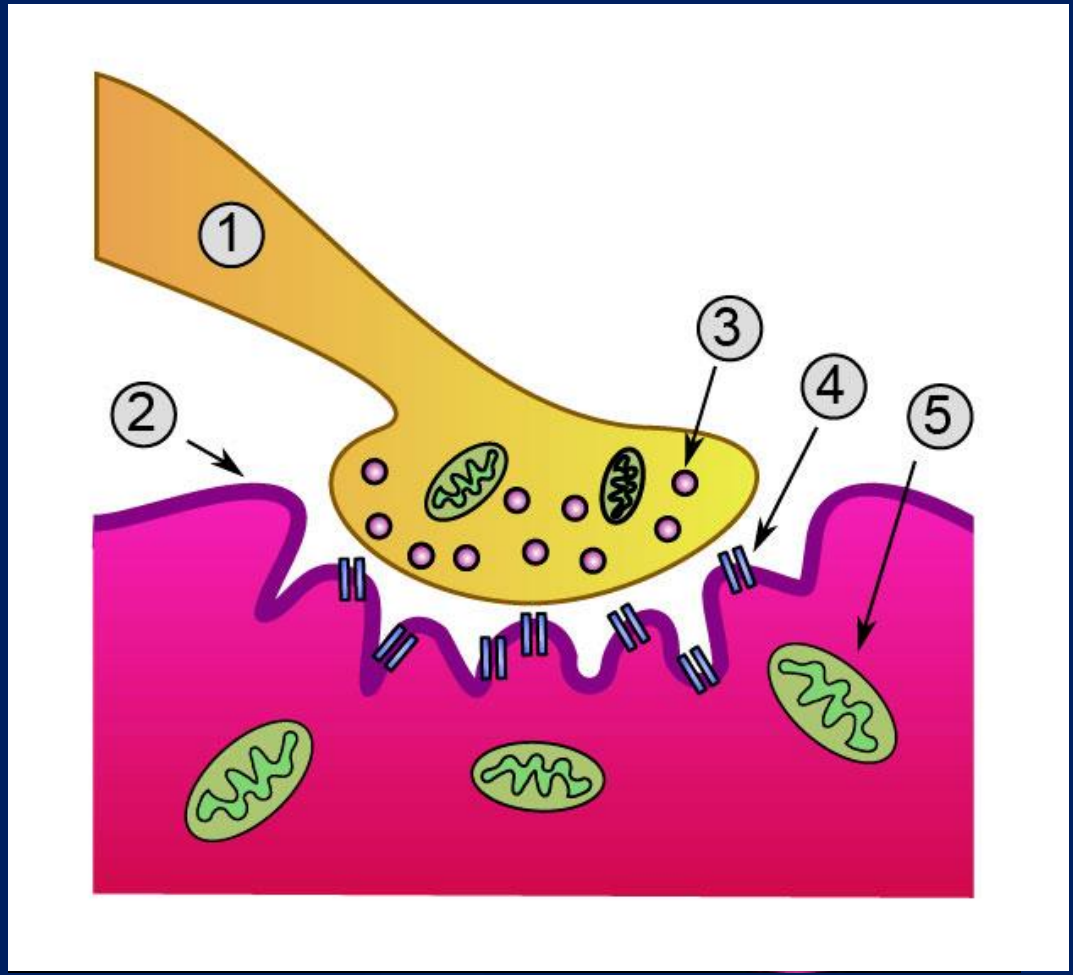
A-a oxygen gradient

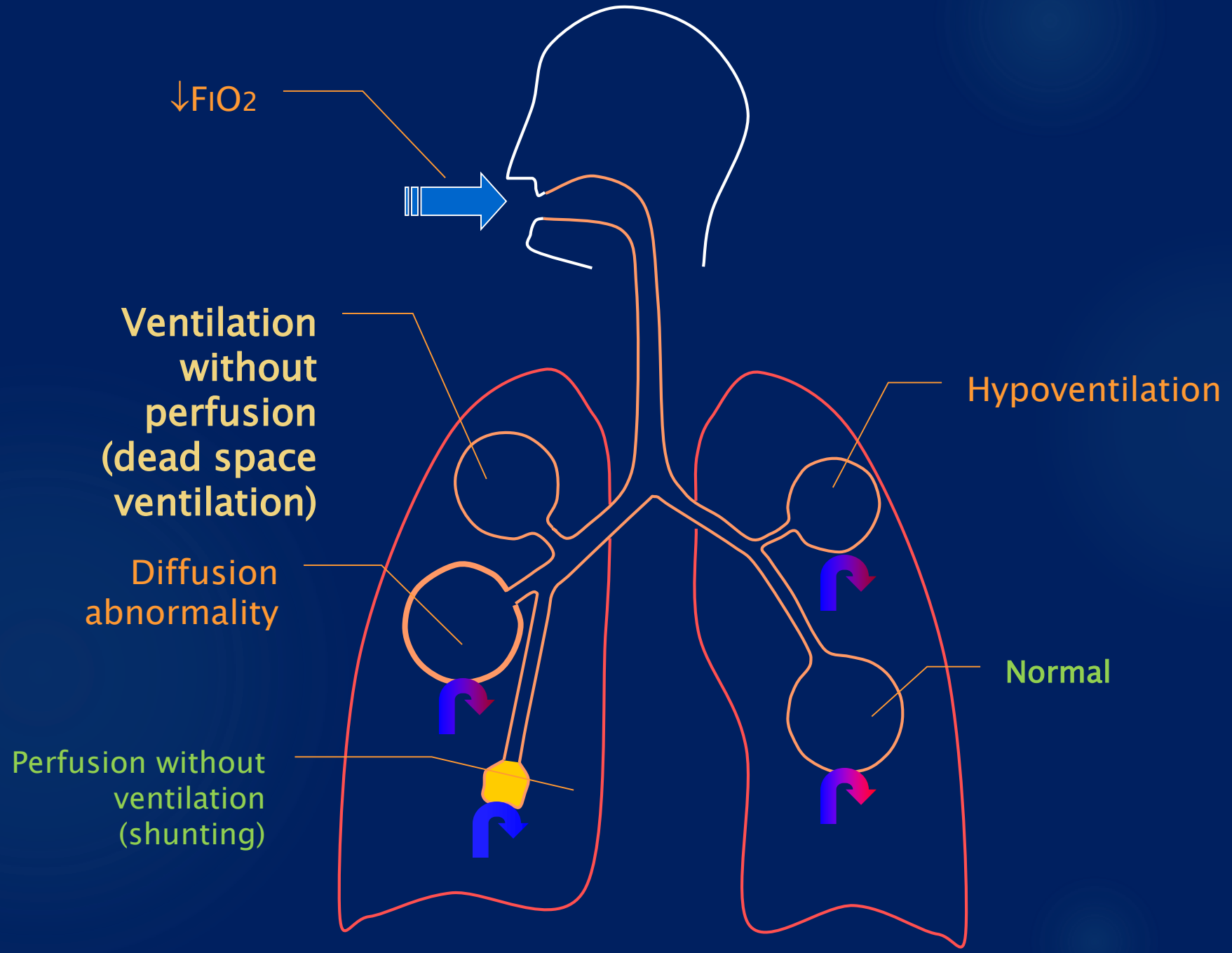
- Air at sea level

- PAo2 = 150 - (Paco2/0.8)
- P(A-a)o2 should be 10 to 15 mm Hg

Abnormalities that cause pure hypoventilation include

- CNS depression
- OHS (Pickwickian) syndrome
- Impaired neural conduction
- Muscular weakness
- Poor chest wall elasticity

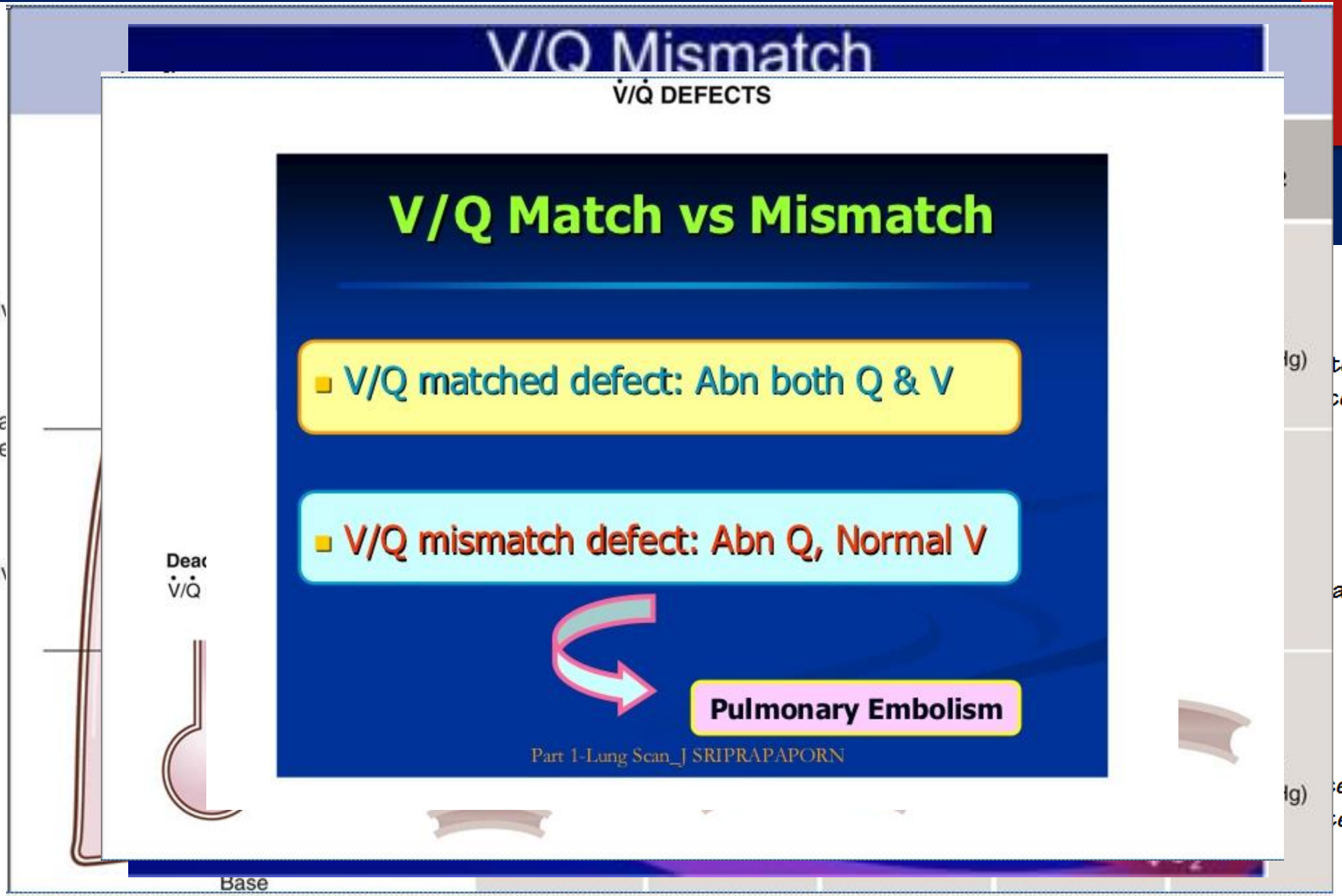



↓FIO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal

V/Q mismatch

- ***imbalance of blood flow and ventilation***
- ***Hypoxemia can be corrected with O2***
- ***increased A-a gradient***

V/Q Mismatch



(From McCance KL, Huether SE, editors: *Pathophysiology: the biologic basis for disease in adults and children*, ed 4, St Louis, 2002, Mosby.)

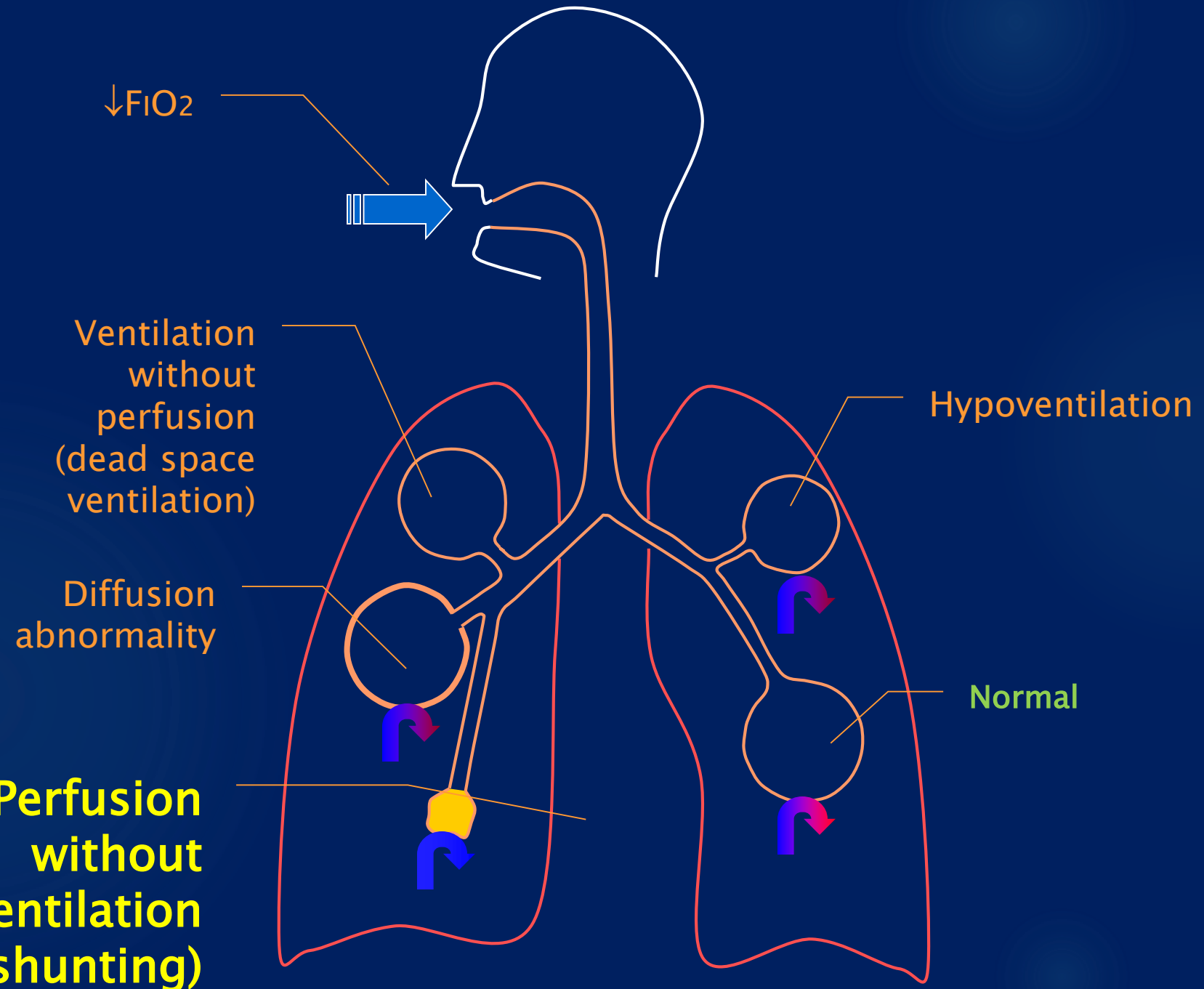
ted Ventilation
ced Perfusion

ation ≅ Perfusion

ed Ventilation
ced Perfusion

V/Q mismatch

- Obstructive lung diseases
- Pulmonary vascular diseases
- Interstitial diseases
- Air-space consolidation
  - Pneumonia
  - Pulmonary edema


↓FIO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal

Right-to-left shunt

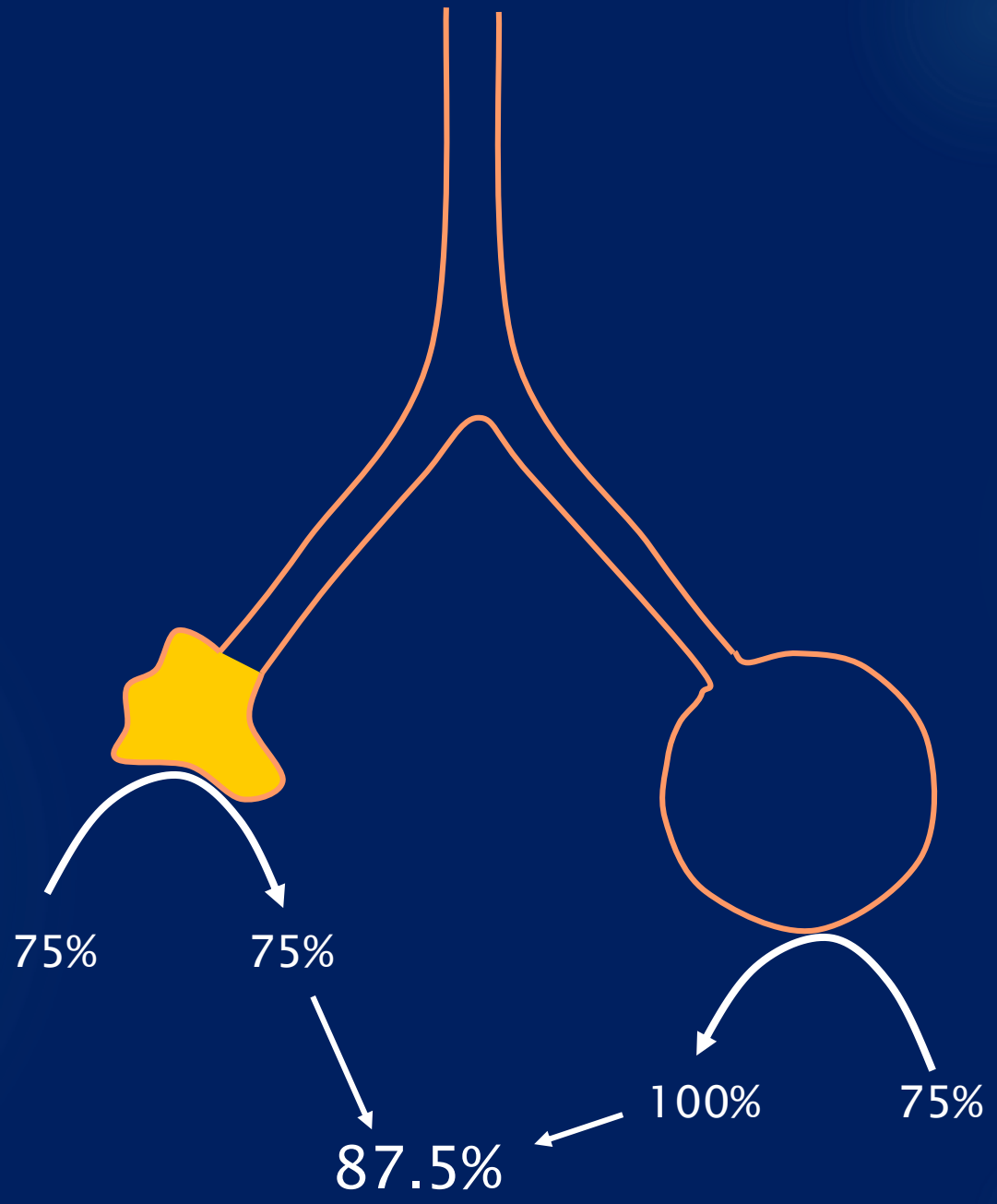
- when blood passes from the right to the left side of the heart
- without being oxygenated

- Anatomic shunts
  - intracardiac shunts
  - pulmonary arteriovenous malformations (AVMs)
  - hepatopulmonary syndrome

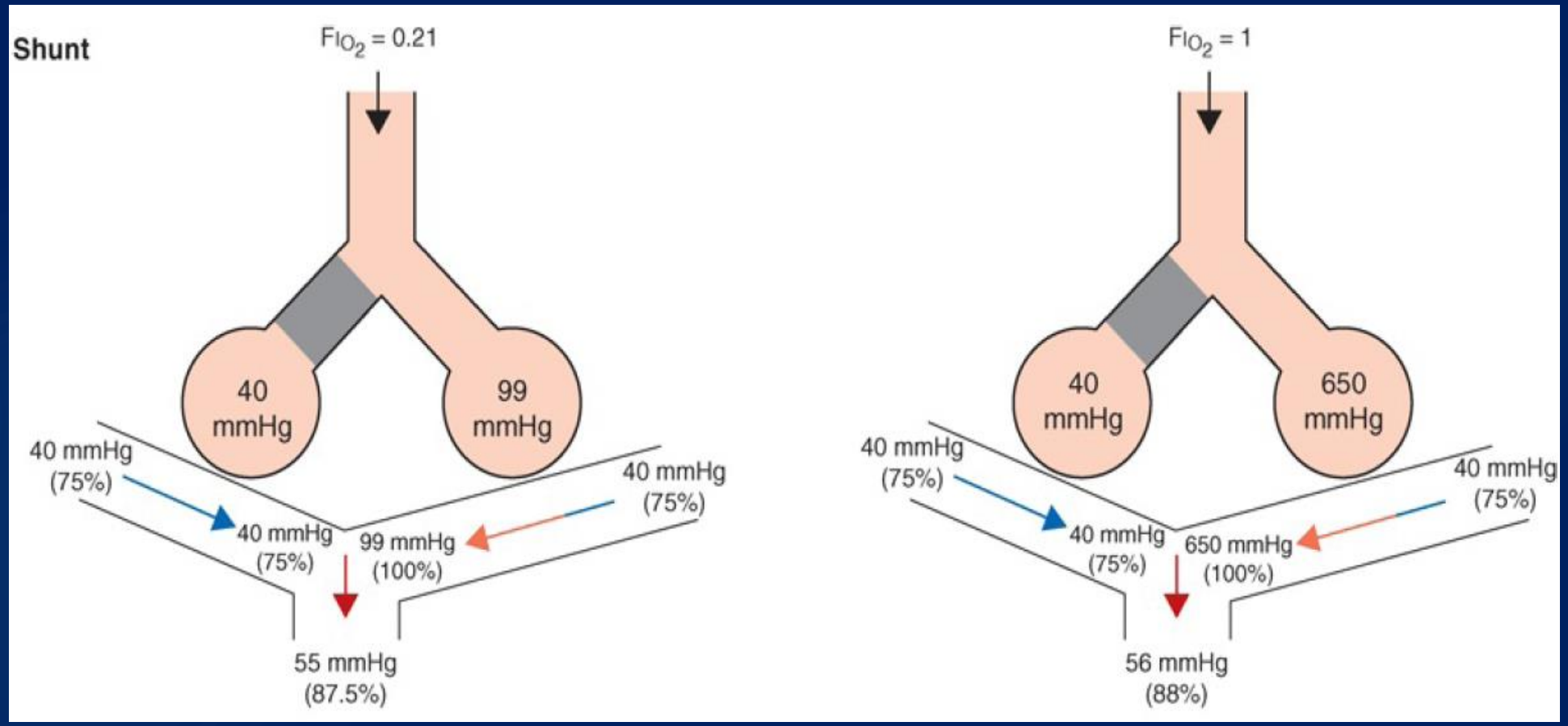
- Physiologic shunts
- atelectasis
- alveolar filling (eg, pneumonia, ARDS)

Difficult to correct with supplemental oxygen

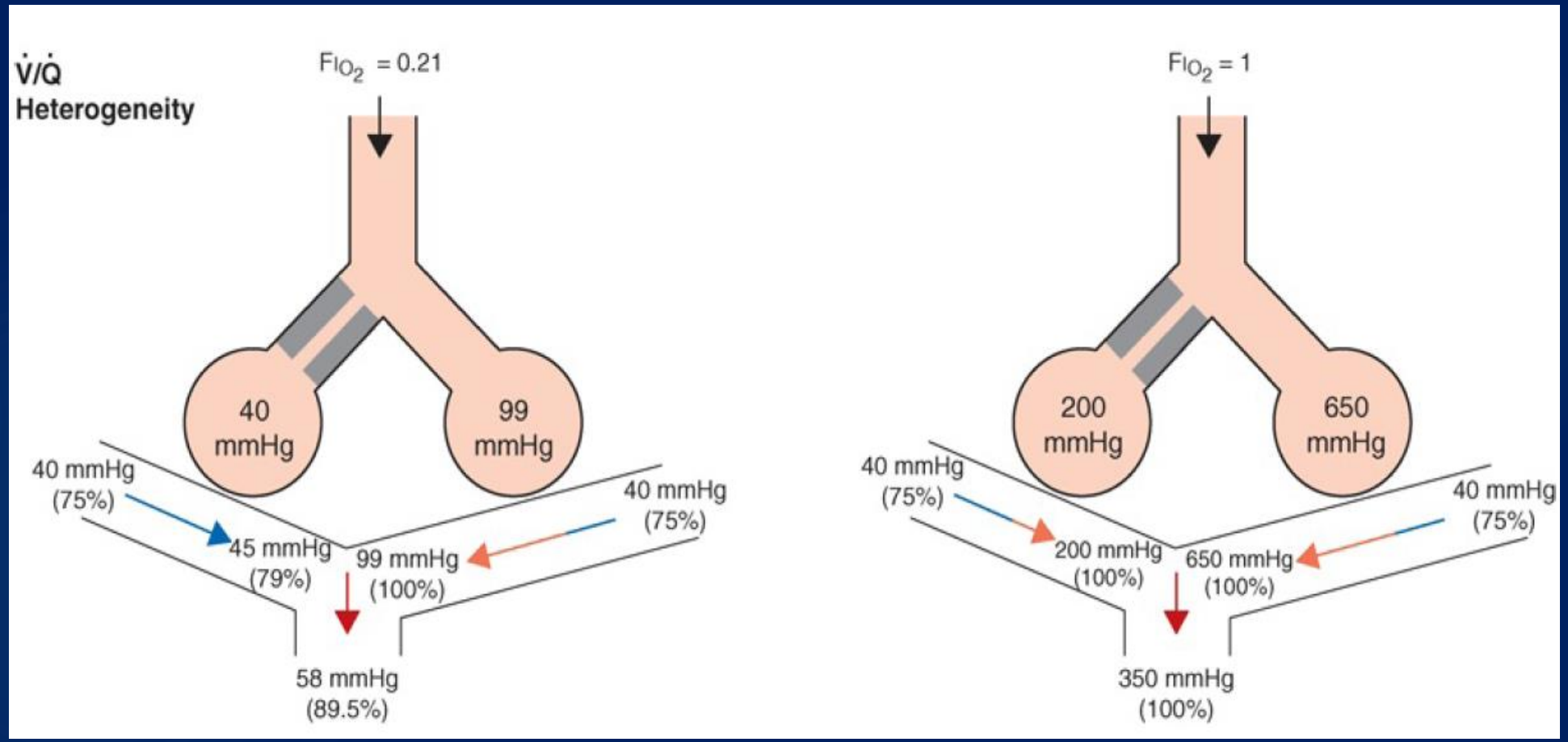
Shunt

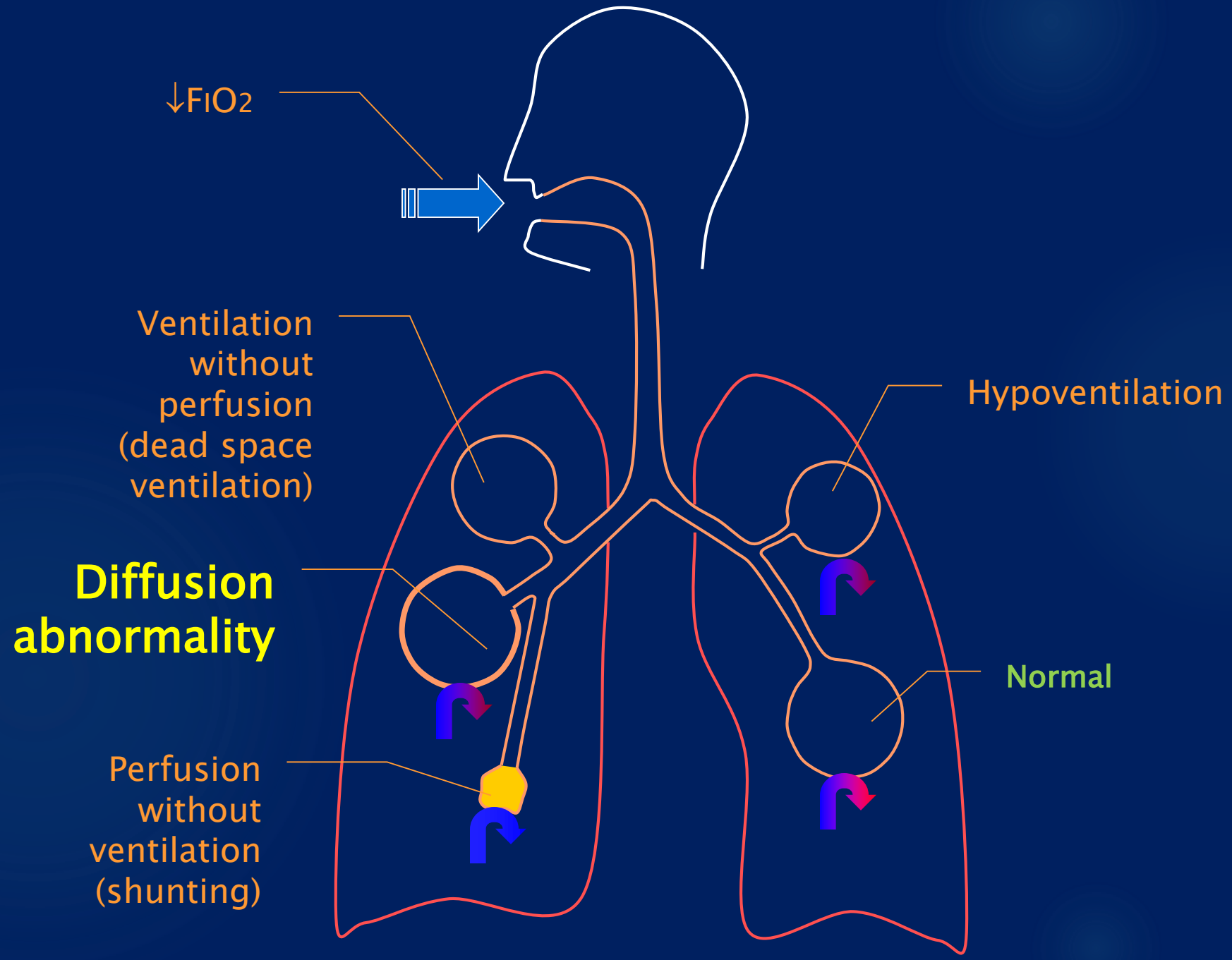


Shunt


Shunt
FIO_2 = 0.21
40 mmHg
99 mmHg
40 mmHg (75%)
40 mmHg (75%)
40 mmHg (75%)
99 mmHg (100%)
55 mmHg (87.5%)
FIO_2 = 1
40 mmHg
650 mmHg
40 mmHg (75%)
40 mmHg (75%)
40 mmHg (75%)
650 mmHg (100%)
56 mmHg (88%)

V/Q mismatch


V̇/Q̇
Heterogeneity
FIO2 = 0.21
40 mmHg
99 mmHg
40 mmHg (75%)
45 mmHg (79%)
99 mmHg (100%)
40 mmHg (75%)
58 mmHg (89.5%)
FIO2 = 1
200 mmHg
650 mmHg
40 mmHg (75%)
200 mmHg (100%)
650 mmHg (100%)
40 mmHg (75%)
350 mmHg (100%)

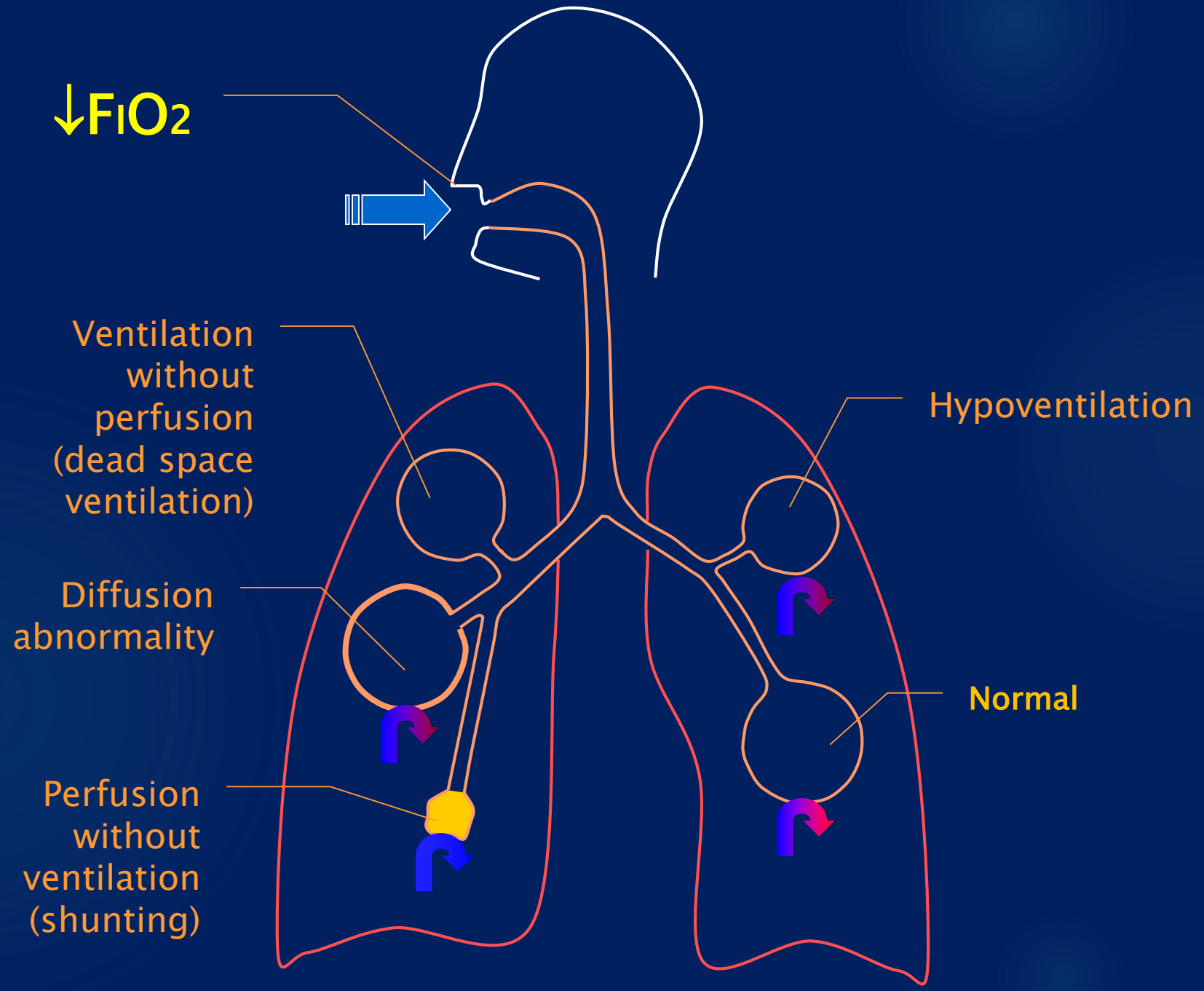

↓FIO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal

Diffusion limitation

- alveolar and/or interstitial inflammation and fibrosis
- interstitial lung disease

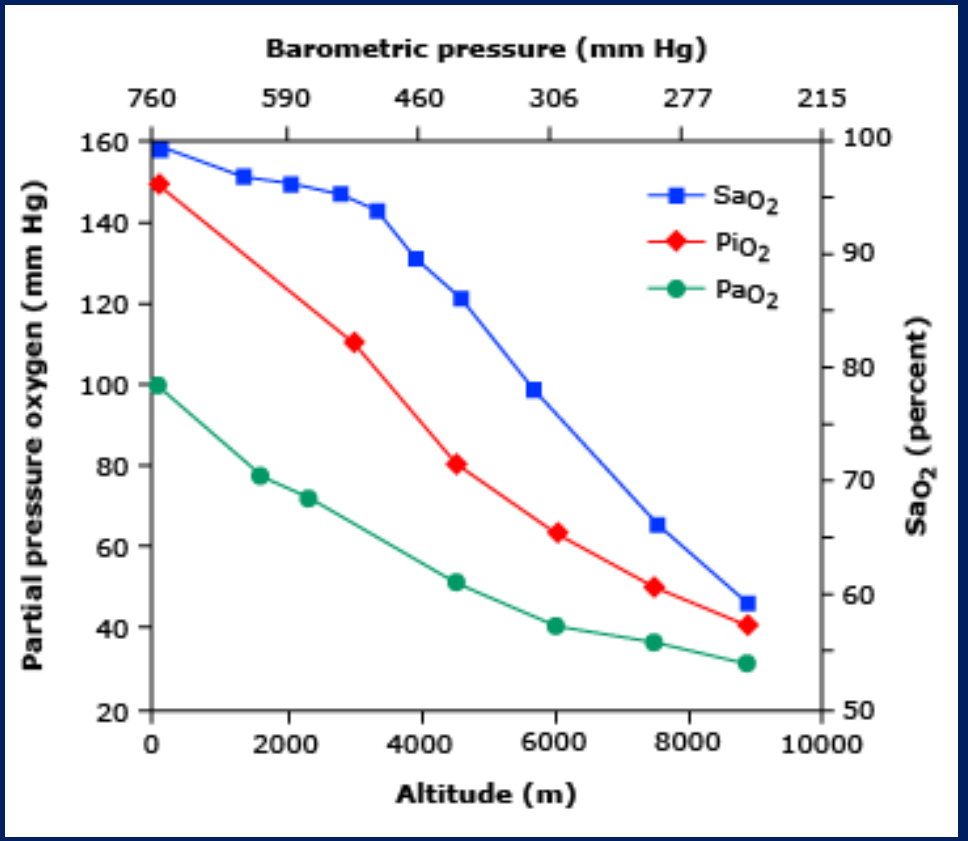
- diffusion limitation usually coexists with V/Q mismatch

- exercise-induced or -exacerbated hypoxemia


↓FiO2
Ventilation without perfusion (dead space ventilation)
Diffusion abnormality
Perfusion without ventilation (shunting)
Hypoventilation
Normal

Reduced inspired oxygen tension

- PiO2 = FiO2 x (Patm - PH2O)
- Reduction of the PiO2 will decrease the PAO2



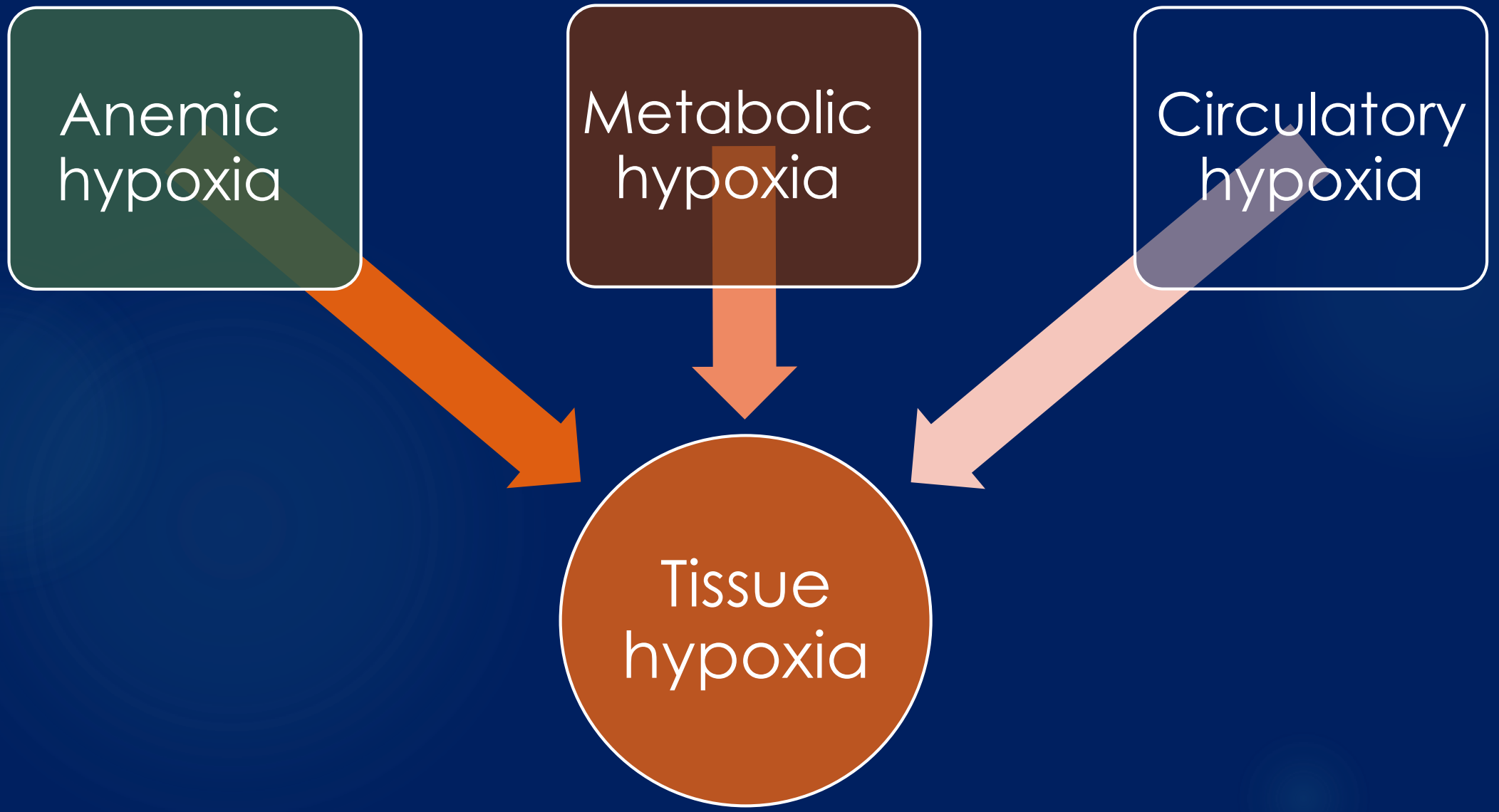
- A reduced PiO2 is most commonly associated with high altitude

Clinical Features and Diagnostic Approach

- Tachypneic
- Tachycardic
- **Cyanosis** of the lips or tongue

- Reduced (deoxygenated) hemoglobin is **> 5 g/100 mL**

Impair oxygen delivery or utilization



Physical examination

- CHF
- Consolidation or effusion
- Pneumothorax

- ***Arterial hypoxemia***

Normal A-aPo2 gradient

Hypoventilation

	pCO_2	pO_2
Hypoventilation	80 mm Hg	40 mm Hg
Normal Ventilation	40 mm Hg	90 mm Hg
Hyperventilation	20 mm Hg	115 mm Hg

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

First description of ARDS

- in 1967 Ashbaugh and colleagues described 12 patients ranging in age from 11 to 48 years who presented with
- **respiratory distress**
- **hypoxemic respiratory failure**
- **patchy bilateral opacities on chest radiographs**

Acute respiratory distress syndrome (ARDS)

- ***clinical syndrome***
- severe dyspnea of rapid onset
- Hypoxemia
- diffuse pulmonary infiltrates

- respiratory failure

ARDS

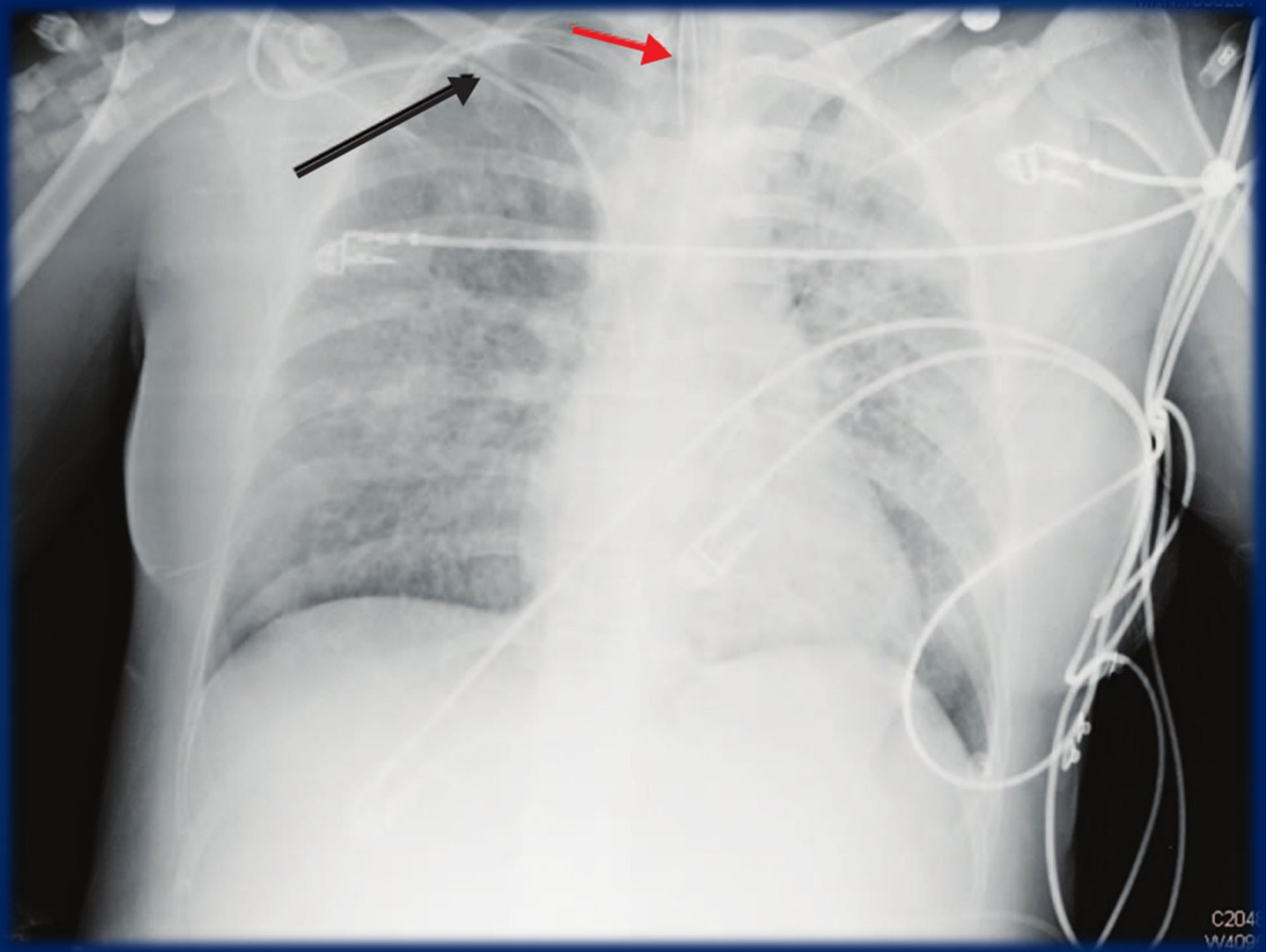
Table 23.2	Clinical Features of ARDS
	1. Acute onset
	2. Bilateral infiltrates on frontal chest x-ray
	3. $PaO_2/FIO_2 \leq 300$ mm Hg†
	4. No evidence of left heart failure or fluid overload
	5. The presence of a predisposing condition

clinical features of ARDS

TABLE 294-2 Diagnostic Criteria for ARDS

SEVERITY: OXYGENATION[a]	ONSET	CHEST RADIOGRAPH	ABSENCE OF LEFT ATRIAL HYPERTENSION
Mild: 200 mmHg < Pao_2/Fio_2 ≤ 300 mmHg *Moderate:* 100 mmHg < Pao_2/Fio_2 ≤ 200 mmHg *Severe:* Pao_2/Fio_2 ≤ 100 mmHg	Acute: Within 1 week of a clinical insult or new or worsening respiratory symptoms.	Bilateral opacities consistent with pulmonary edema not fully explained by effusions, lobar/lung collapse, or nodules	Hydrostatic edema is not the primary cause of respiratory failure. If no ARDS risk factor is present, then some objective evaluation is required (e.g., echocardiography) to rule out hydrostatic edema

Three categories based on the degrees of hypoxemia




ARDS

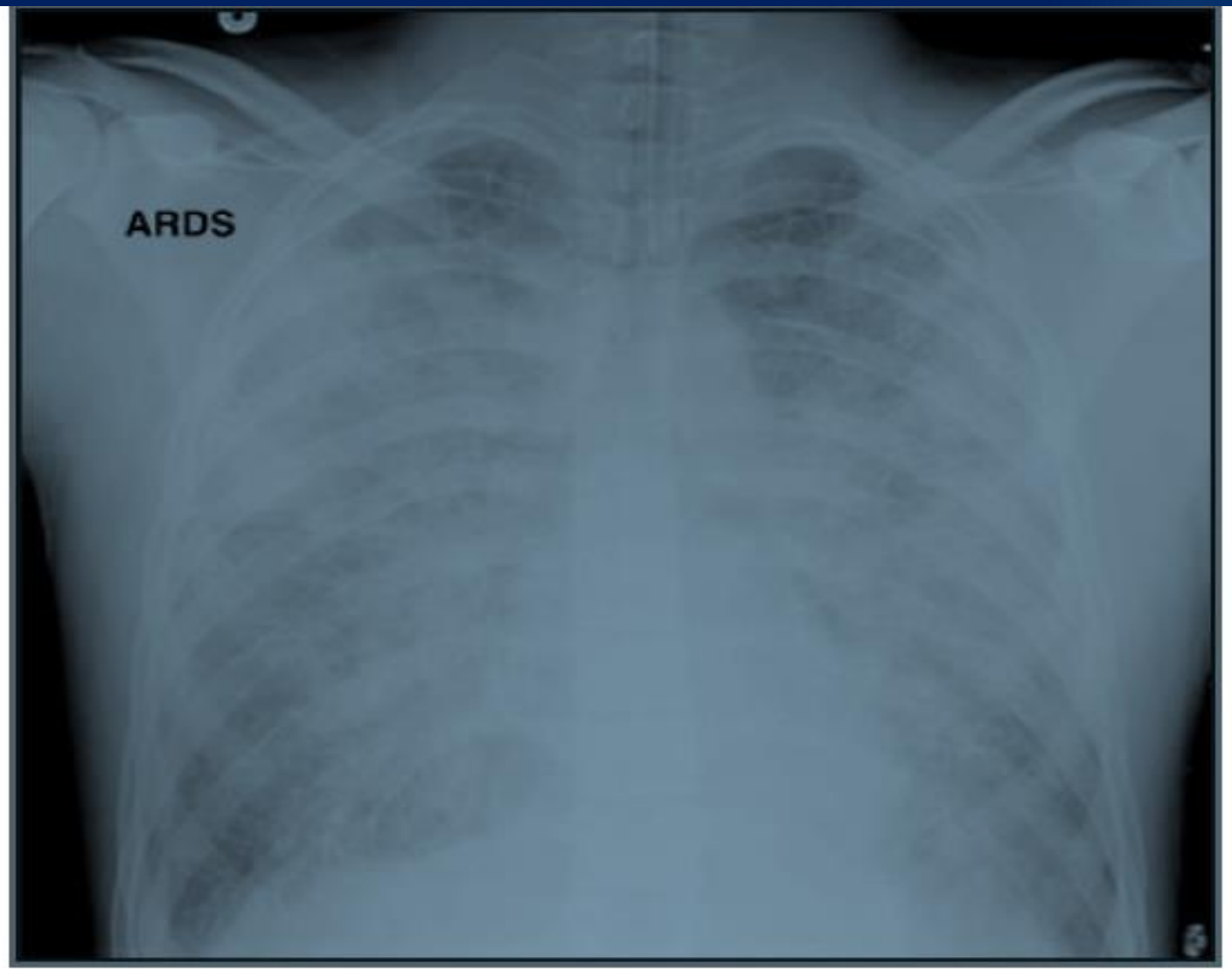
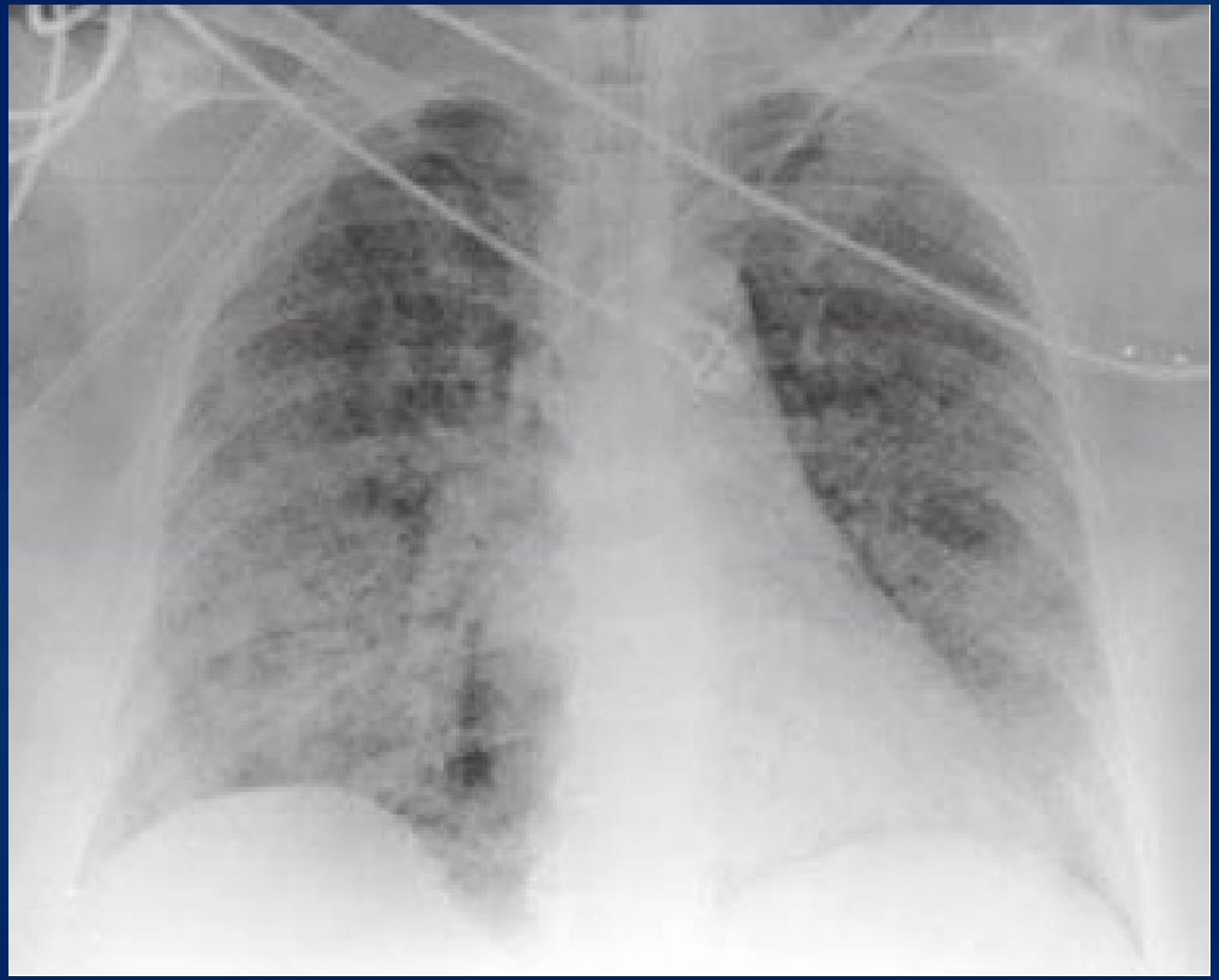
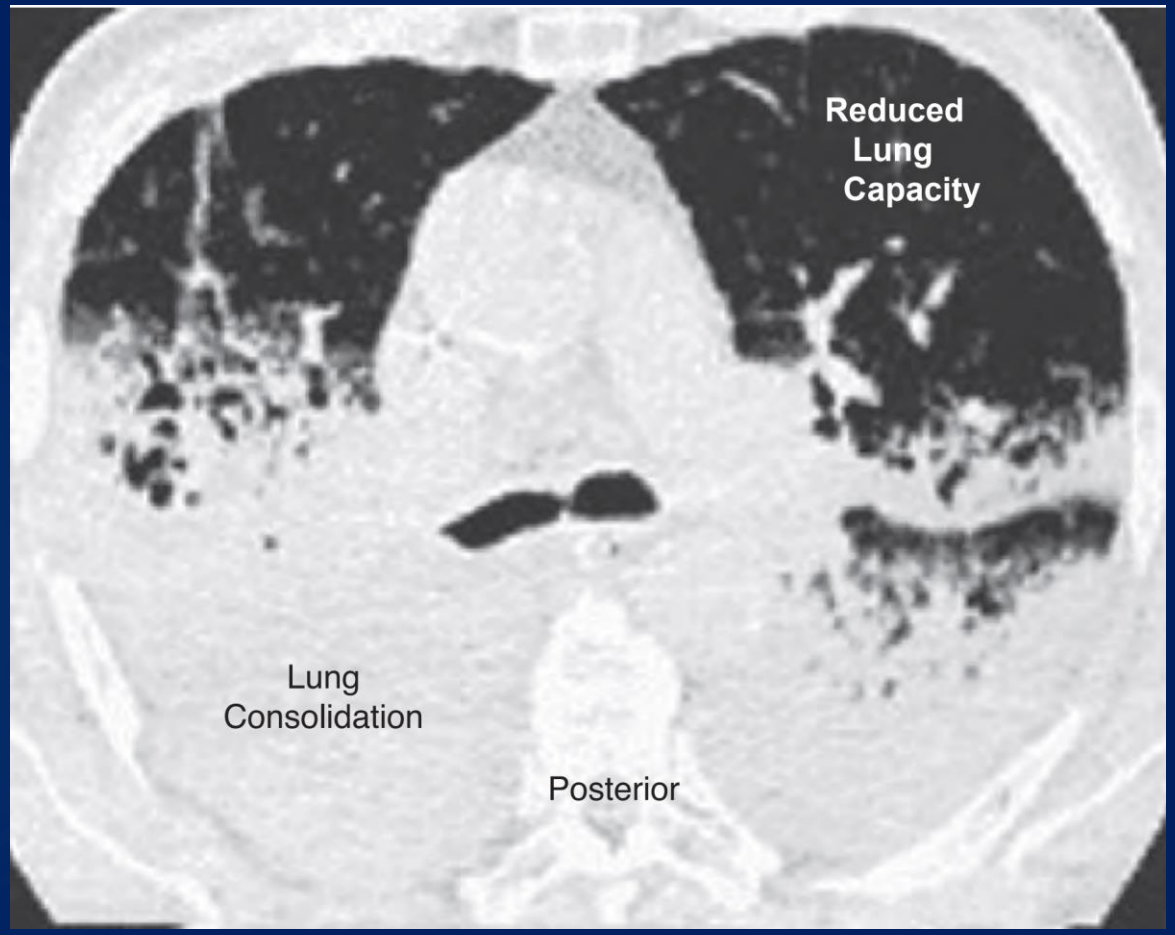


FIGURE 23.2 Portable chest x-ray showing the classic radiographic appearance of ARDS. The infiltrate has a finely granular or "ground glass" appearance, and is evenly distributed throughout both lungs, with a relative sparing of the lung bases. There is no evidence of a pleural effusion.




Reduced
Lung
Capacity
Lung
Consolidation
Posterior

Acute respiratory distress syndrome (ARDS)

- ***Diffuse lung injury***
  - many underlying medical and surgical disorders

- lung injury
  - Direct
    - toxic inhalation
  - Indirect
    - Sepsis

TABLE 294-1 Clinical Disorders Commonly Associated with ARDS

DIRECT LUNG INJURY	INDIRECT LUNG INJURY
Pneumonia	Sepsis
Aspiration of gastric contents	Severe trauma
Pulmonary contusion	Multiple bone fractures
Near-drowning	Flail chest
Toxic inhalation injury	Head trauma
	Burns
	Multiple transfusions
	Drug overdose
	Pancreatitis
	Postcardiopulmonary bypass

ARDS

- Acute onset
- Bilateral opacities
- PAWP <=18 mm Hg

  (absence of clinical evidence of LAH)
- Hypoxemia Po2/Fio2

ARDS

TABLE 322-2 DIAGNOSTIC CRITERIA FOR ARDS

Severity: Oxygenation	Onset	Chest Radiograph	Absence of Left Atrial Hypertension
Mild: 200 mmHg < Pao_2/FIO_2 ≤ 300 mmHg *Moderate:* 100 mmHg < Pao_2/FIO_2 ≤ 200 mmHg *Severe:* Pao_2/FIO_2 ≤ 100 mmHg	Acute	Bilateral alveolar or interstitial infiltrates	PCWP ≤18 mmHg *or* no clinical evidence of increased left atrial pressure

ARDS

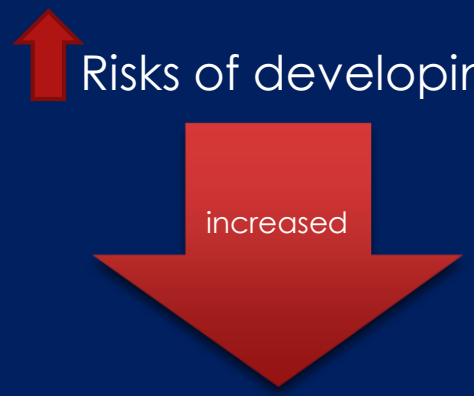
- annual incidence 60 cases/100,000 population
- 10% of all ICU admissions

ETIOLOGY

- many medical and surgical illnesses
  - pneumonia and sepsis (~40–60%)
  - aspiration of gastric contents
  - Trauma
  - multiple transfusions
  - drug overdose

ETIOLOGY

- many medical and surgical illnesses
  - pneumonia and sepsis (~40–60%)
  - aspiration of gastric contents
  - Trauma
    - Pulmonary contusion
    - multiple bone fractures
    - chest wall trauma/flail chest
    - head trauma
    - near-drowning
    - toxic inhalation
    - Burns
  - multiple transfusions
  - drug overdose



> 1 predisposing medical or surgical condition

Lack of Specificity

- Many of the diagnostic criteria for ARDS

- ***misdiagnosis*** → ***Pneumonia***
  Hydrostatic pulmonary edema

Other Risk Factors

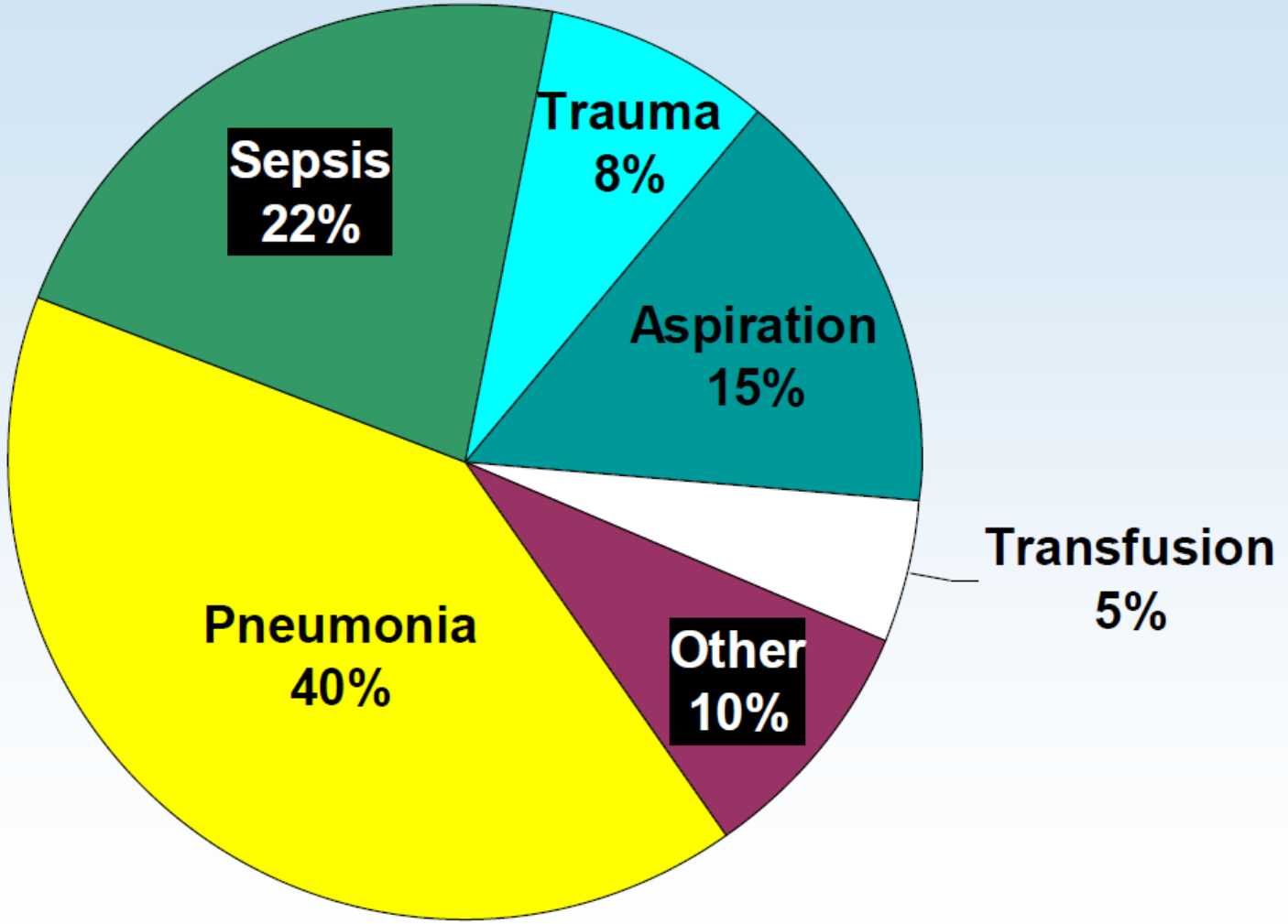
- ***Older age***
- ***Chronic alcohol abuse***
- ***Metabolic acidosis***
- ***Pancreatitis***
- ***Severity of critical illness***
- Trauma
  - APACHE II score ≥16
    - 2.5-fold increased risk of developing ARDS

Diabetes hyperglycemia is known to impair neutrophil function

ARDS

- **Direct injury to the lung**
  - aspiration pneumonia
  - smoke inhalation
  - near-drowning
- **systemic injury**
  - Trauma
  - Surgery
  - Sepsis
  - Burns
  - Long bone fractures
  - Pancreatitis
  - Uremia
  - transfusion therapy
  - Shock
  - drug intoxication
  - cardiopulmonary bypass

NIH-NHLBI ARDS Network
Cause of Lung Injury



NHLBI ARDS Clinical Trials Network. N Engl J Med. 2004.

Cigarette smoke

- ***active or passive exposure to cigarette smoke***

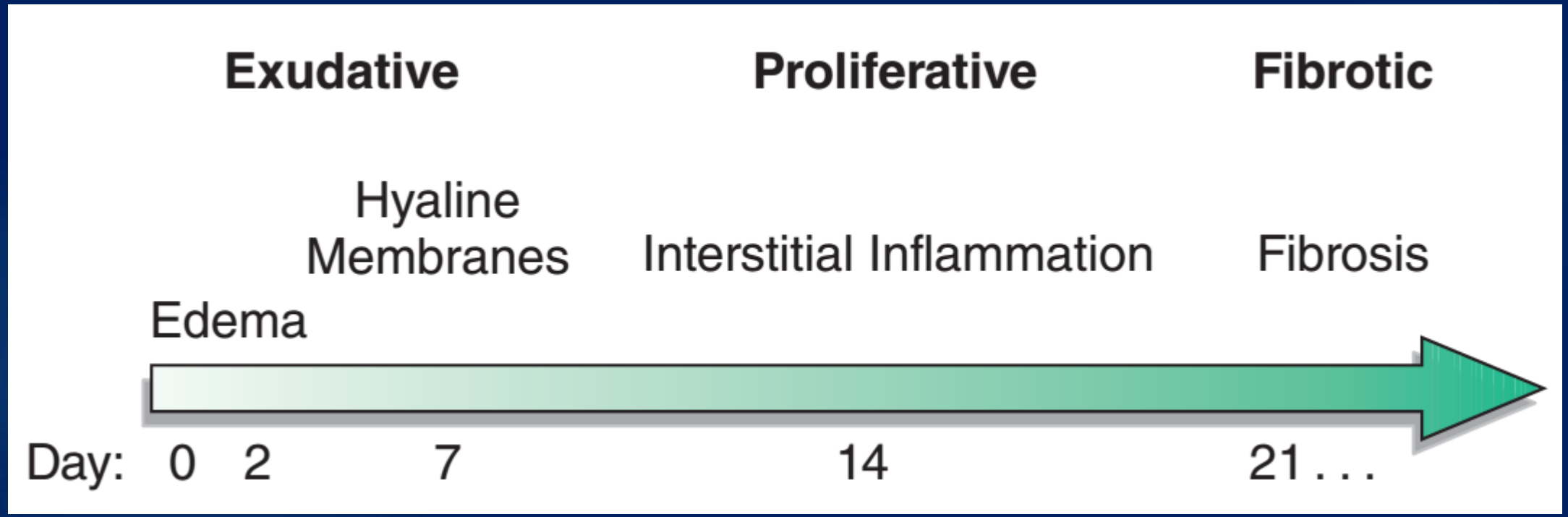
development of ARDS after trauma

Overview of Pathophysiology in ARDS

- ***edema fluid that fills the alveoli is exudative***
- ***alveolar-capillary barrier exhibits increased permeability***
- **leakage of protein-rich fluid into the air spaces**

- ↓ respiratory system compliance
- Right -to-left shunting
- profound hypoxemia
- $PaCO_2$ is generally within the normal range
- ↑ Dead space ventilation & ↑ minute ventilation

Time course for the development and resolution of ARDS



Pathology

- Exudative phase 5 -7 days
- Proliferative phase Days 7-21
- Fibrotic phase > 21 days
- Recovery phase > 7-21 days

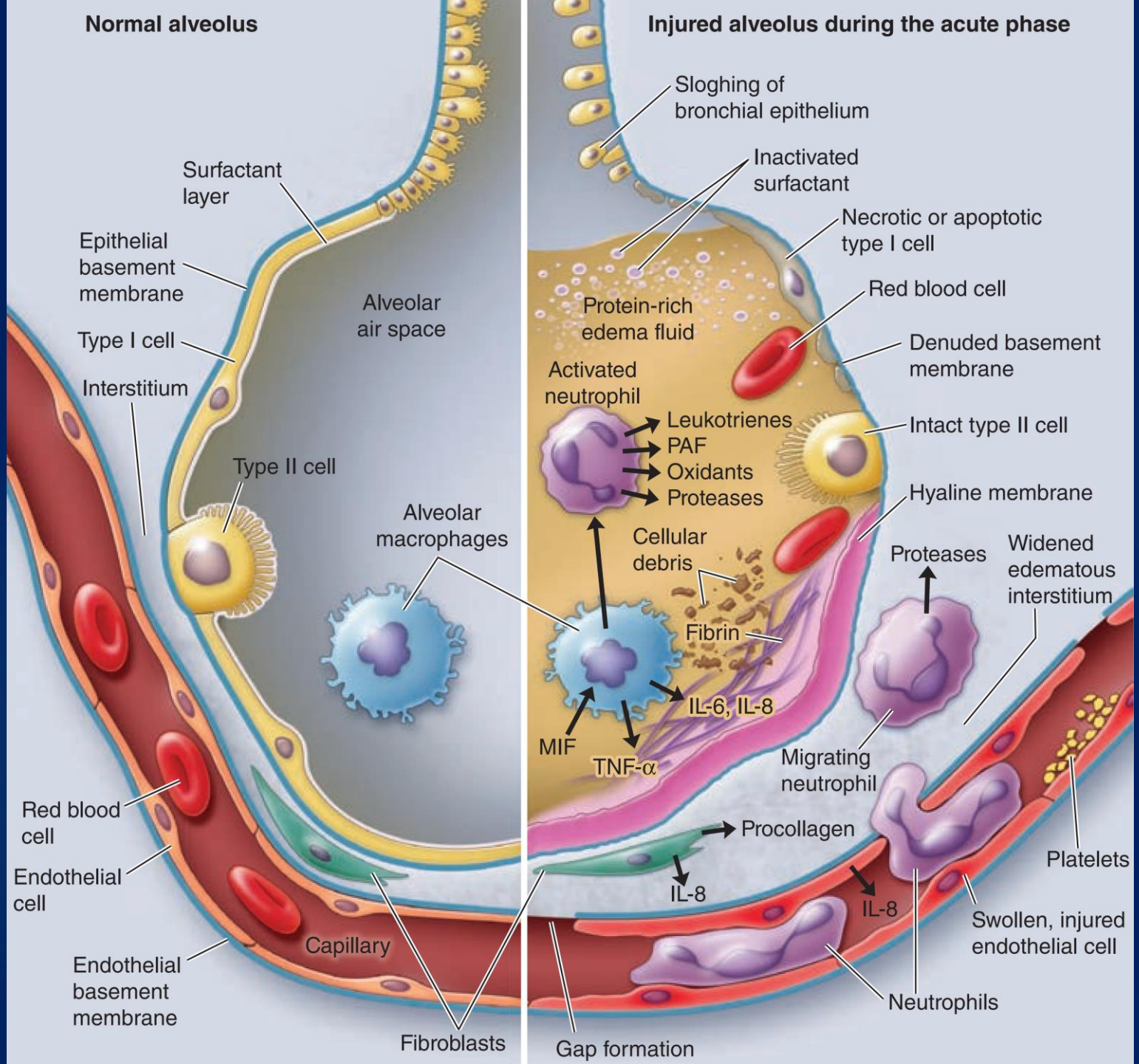
Pathology

- ***Exudative phase 5 -7 days***
- Proliferative phase Days 7-21
- Fibrotic phase > 21 days
- Recovery phase > 7-21 days

Exudative phase

- ***DAD type I pneumocytes (alveolar epithelial cells)***
- Increased permeability
  - Influx of protein rich fluid into alveoli
- enhanced cytokine production
  - IL1, IL 8, TNF-a ,LT B4
- recruitment of leukocytes (especially neutrophils)
- dysfunctional pulmonary surfactant
- vascular obliteration by microthrombi
- fibrocellular proliferation

Exudative phase of ARDS


Normal alveolus
Surfactant layer
Epithelial basement membrane
Type I cell
Interstitium
Alveolar air space
Type II cell
Alveolar macrophages
Red blood cell
Endothelial cell
Capillary
Endothelial basement membrane
Fibroblasts
Injured alveolus during the acute phase
Sloghing of bronchial epithelium
Inactivated surfactant
Necrotic or apoptotic type I cell
Red blood cell
Protein-rich edema fluid
Denuded basement membrane
Activated neutrophil
Leukotrienes
PAF
Oxidants
Proteases
Intact type II cell
Hyaline membrane
Cellular debris
Proteases
Widened edematous interstitium
Fibrin
IL-6, IL-8
MIF
TNF-α
Migrating neutrophil
Procollagen
IL-8
IL-8
Platelets
Swollen, injured endothelial cell
Neutrophils
Gap formation

If no ARDS risk factor is present

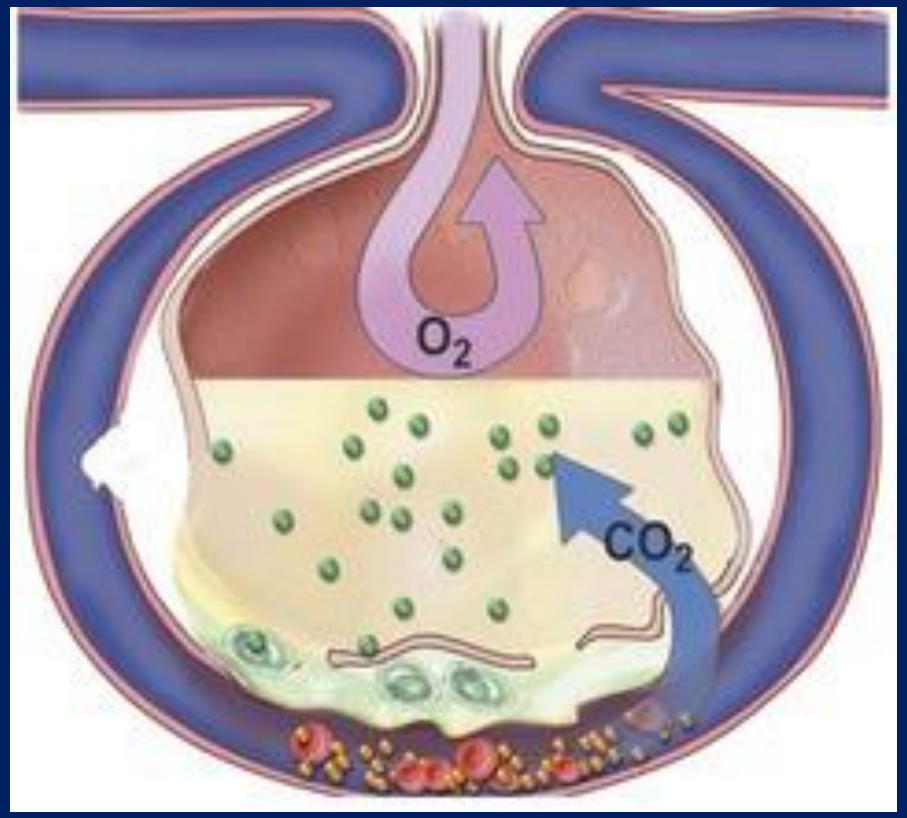
- some objective evaluation is required (e.g., echocardiography)

- to exclude a cardiac etiology for hydrostatic edema

Exudative Phase

- First 7 days of illness after exposure
- 12–36 hours after the initial insult

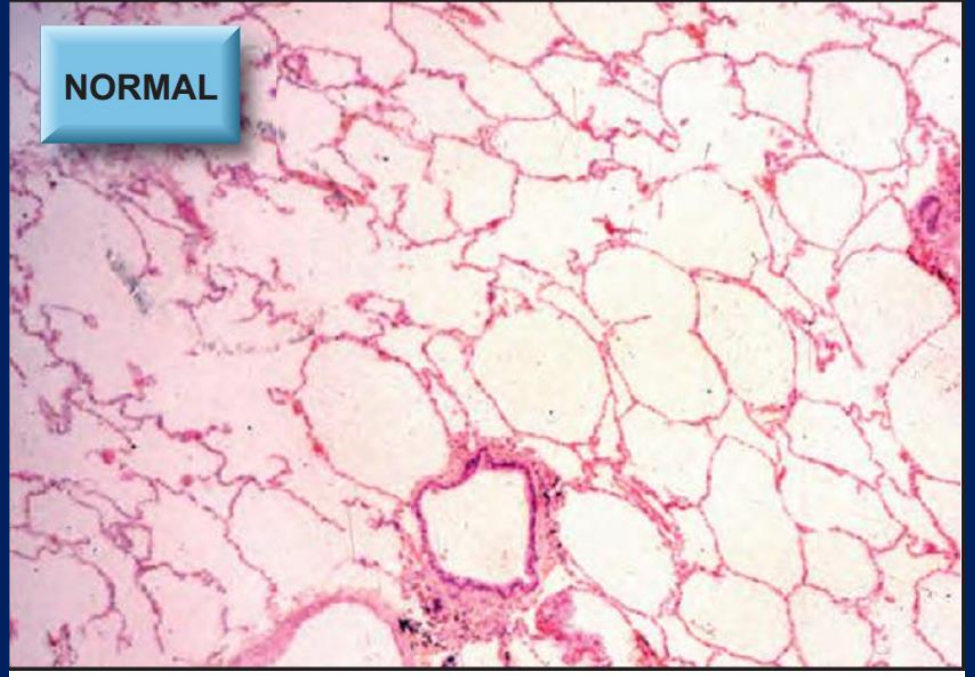
- **Dyspnea**
  - Rapid shallow breathing
  - Inability to get enough air
- **Tachypnea**
  - Increased work of breathing
  - Respiratory fatigue
  - Respiratory failure

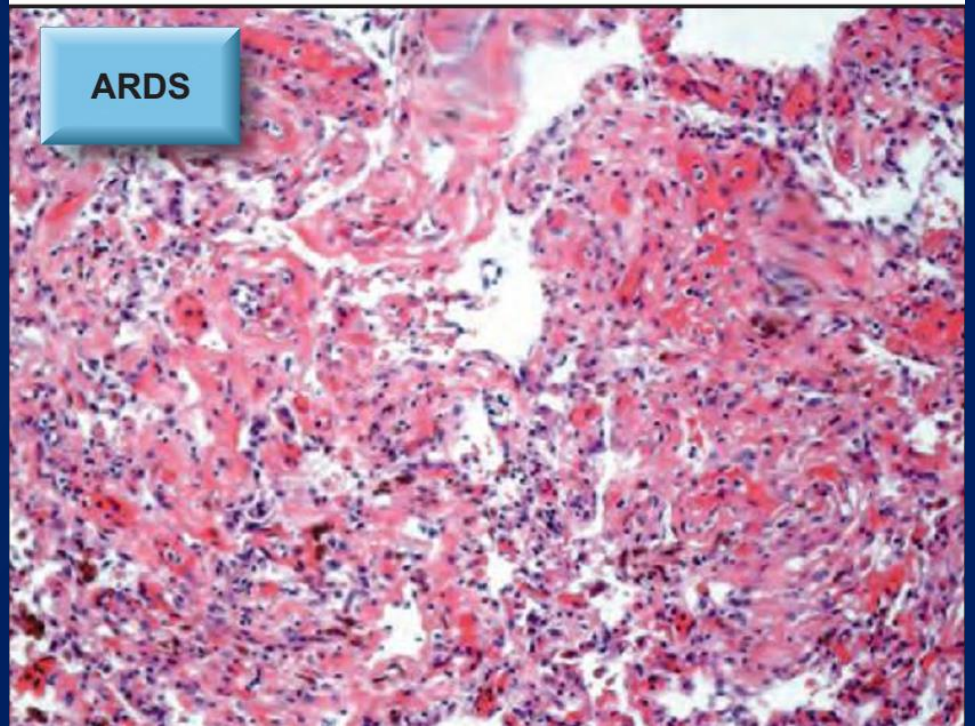


Differential diagnosis of ARDS

- cardiogenic pulmonary edema
- bilateral pneumonia
- alveolar hemorrhage
- acute interstitial lung diseases (AIP)
- acute immunologic injury (HP)
- toxin injury (radiation pneumonitis)
- neurogenic pulmonary edema

ARDS





Pathology

- Exudative phase 5 -7 days
- ***Proliferative phase Days 7-21***
- Fibrotic phase > 21 days
- Recovery phase > 7-21 days

Proliferative phase

- ***Most patients recover rapidly***
- ***many patients still experience***
  - Dyspnea
  - Tachypnea
  - Hypoxemia
- ***Some patients develop progressive lung injury***
  - early changes of pulmonary fibrosis
- ***Shift from neutrophil- to lymphocyte-predominant***
- ***type II pneumocytes proliferate***

Pathology

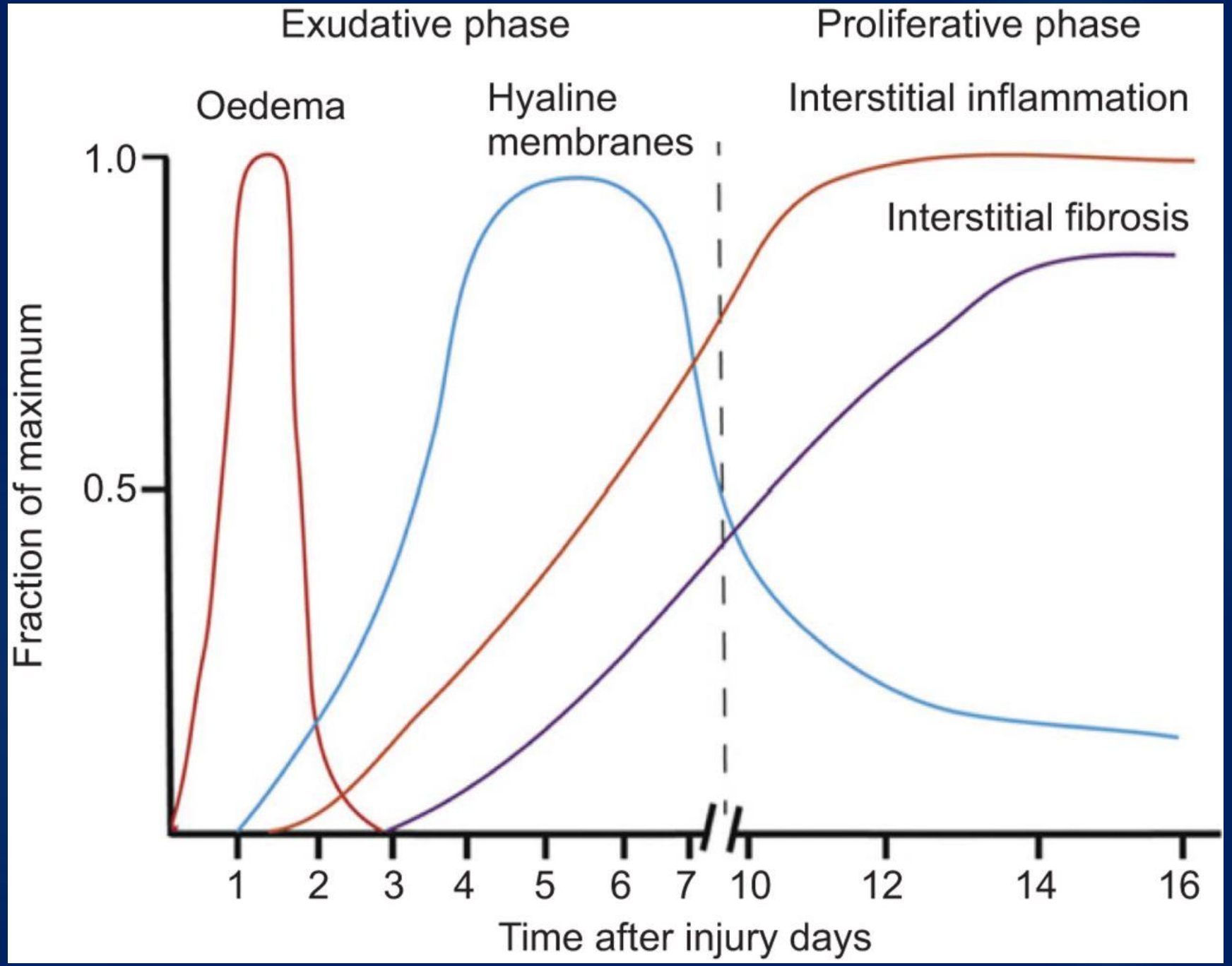
- Exudative phase 5 -7 days
- Proliferative phase Days 7-21
- ***Fibrotic phase > 21 days***
- Recovery phase > 7-21 days

Fibrotic phase 3–4 weeks after

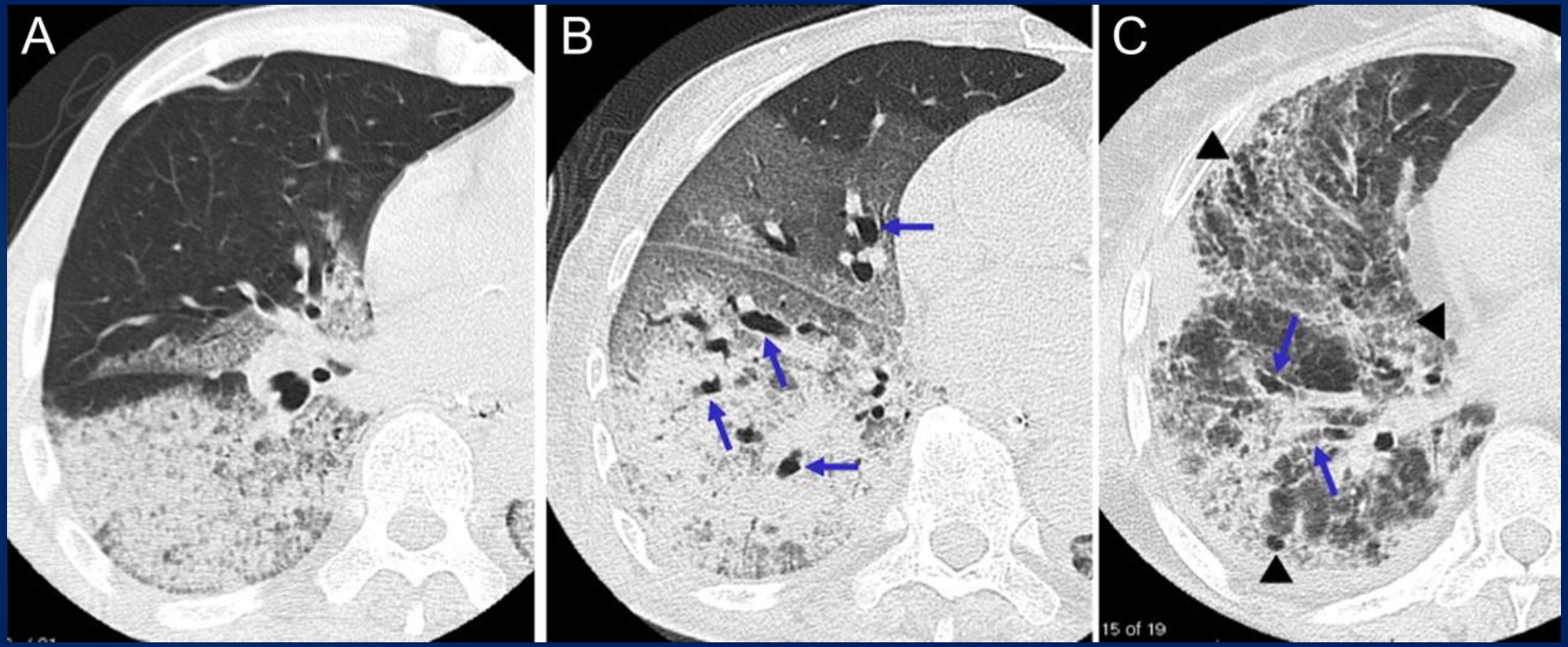
- loss of alveolar structure integrity
- pulmonary fibrosis
  - in a subset of patients with persistent > 3 weeks ARDS

Fibrotic Phase

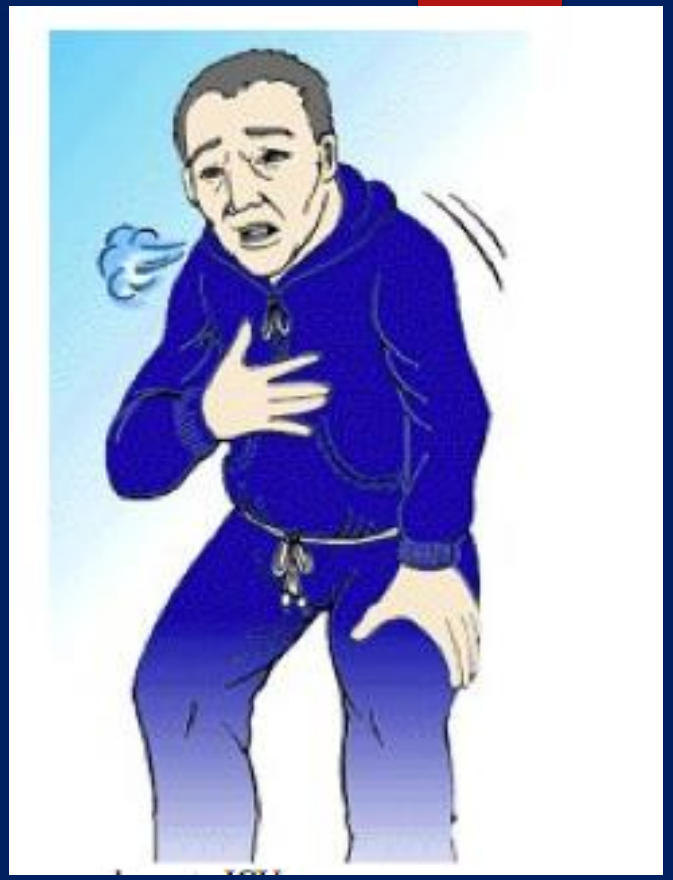
- Interstitial fibrosis
  - long-term support on MV and/or supplemental oxygen.
- emphysema-like changes, with large bullae
  - Marked disruption of acinar architecture
- progressive vascular occlusion and pulmonary hypertension
  - Intimal fibroproliferation in the pulmonary microcirculation
- Pneumothorax
- Lung compliance ↓
- Pulmonary dead space ↑


Exudative phase
Proliferative phase
Oedema
Hyaline membranes
Interstitial inflammation
Interstitial fibrosis
1.0
0.5
Fraction of maximum
1
2
3
4
5
6
7
10
12
14
16
Time after injury days

ARDS



Clinical presentation of an ARDS patient



- hypoxia
- cyanosis
- tachypnea
- tachycardia
- respiratory alkalosis
- respiratory acidosis

- *** this is usually a diagnosis of exclusion
- rule out a couple of things like pneumonia, HF exacerbation

Clinical presentation

- Dyspnea
- Cyanosis
- Tachypnea
- Tachycardia
- Diaphoresis
- Diffuse crackles

Ventila ry

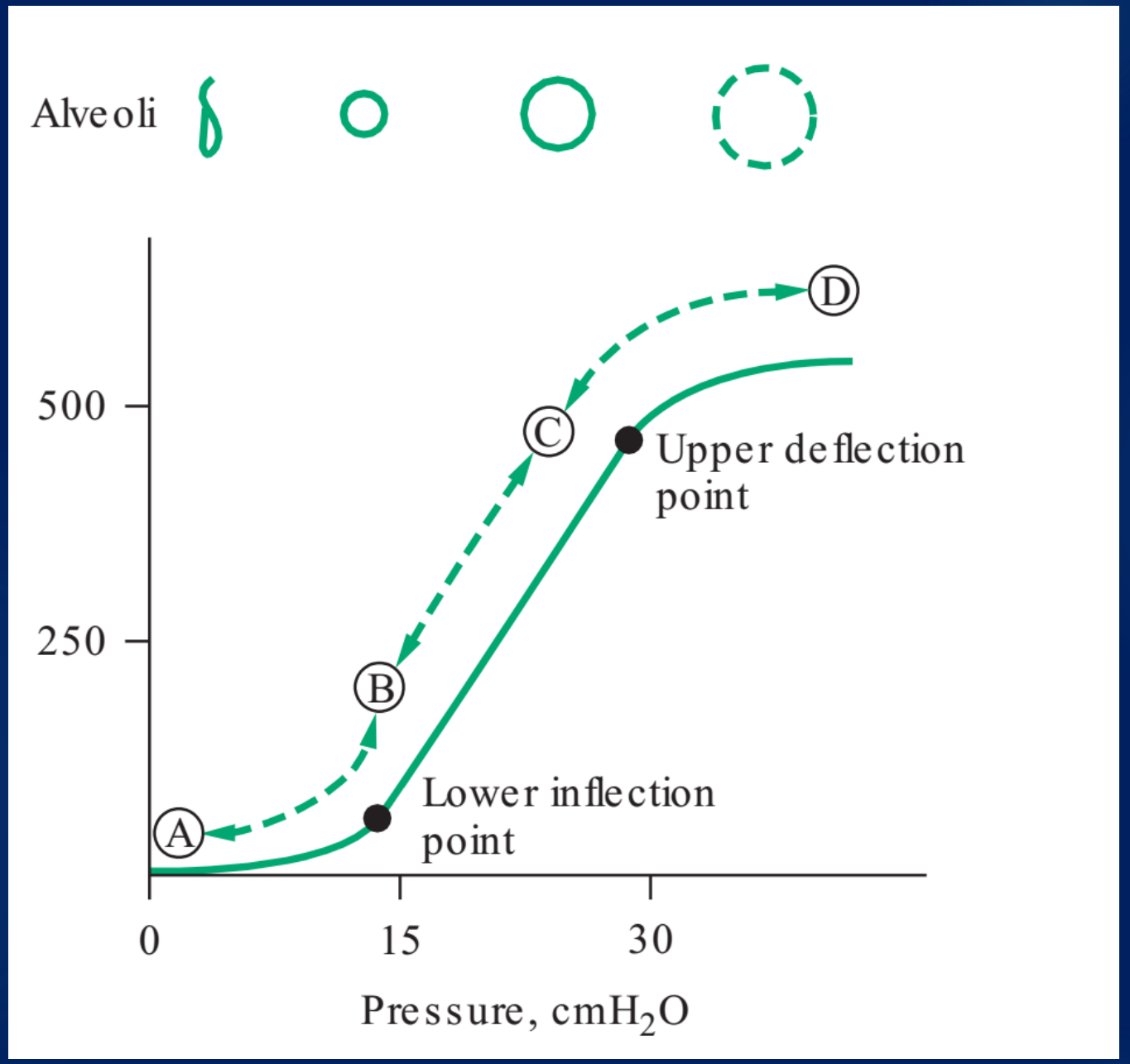
- mechan
- excessiv

- Pneumo
- pneumo
- subcuta

Mechanical ventilation can induce pulmonary edema by causing increases in both epithelial and endothelial permeability

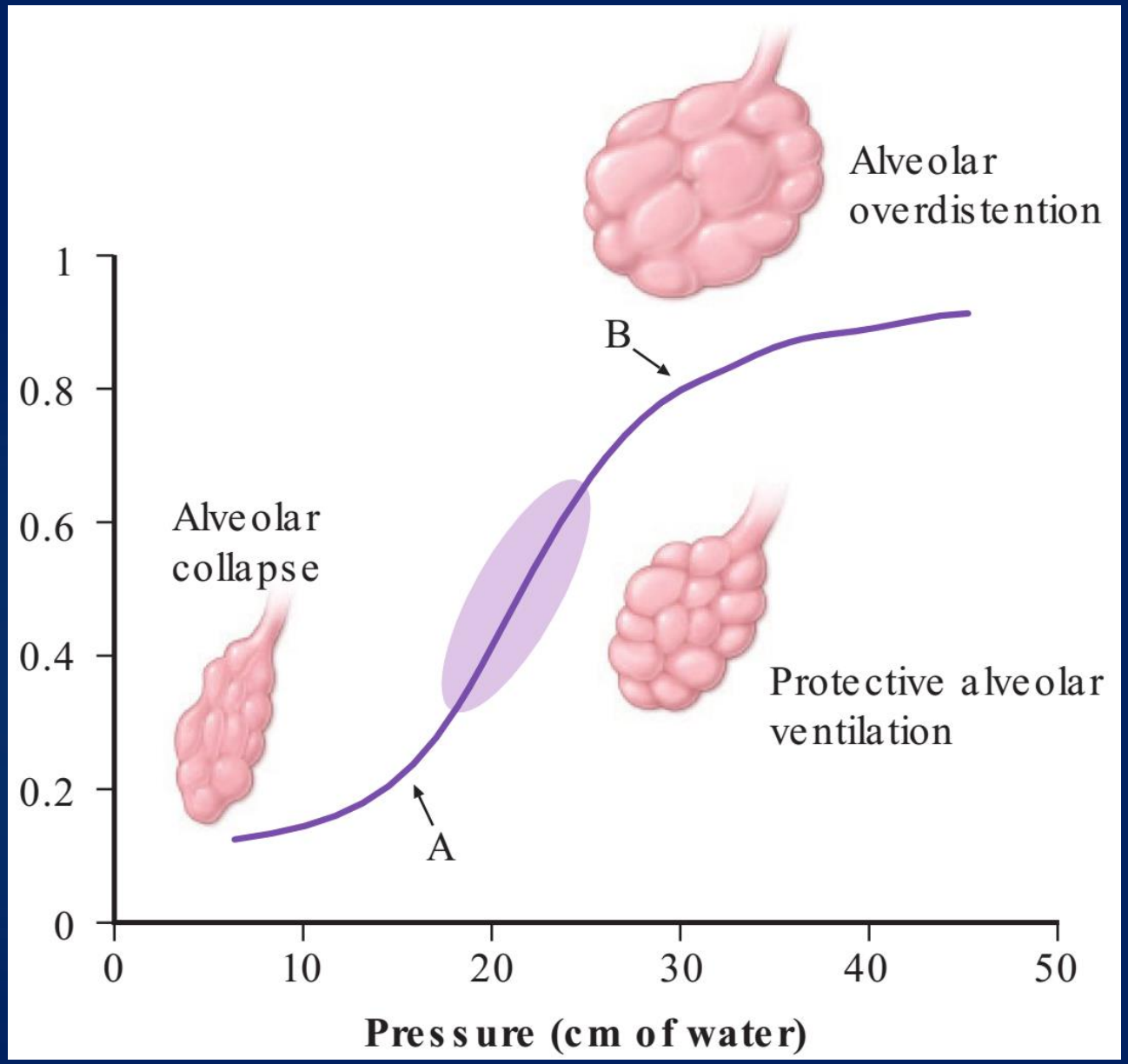

Elevation in cytokine levels

ARDS

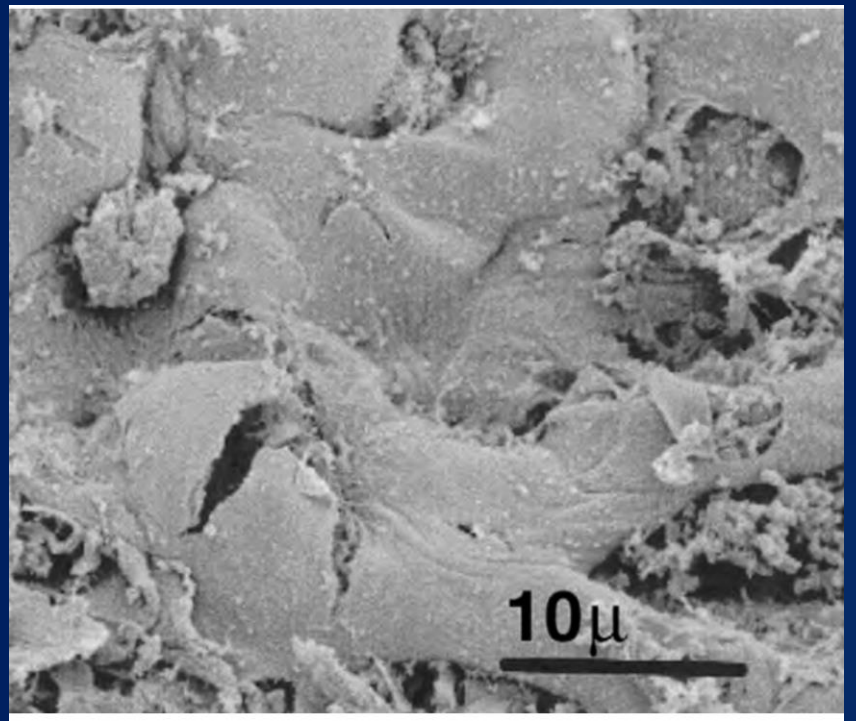


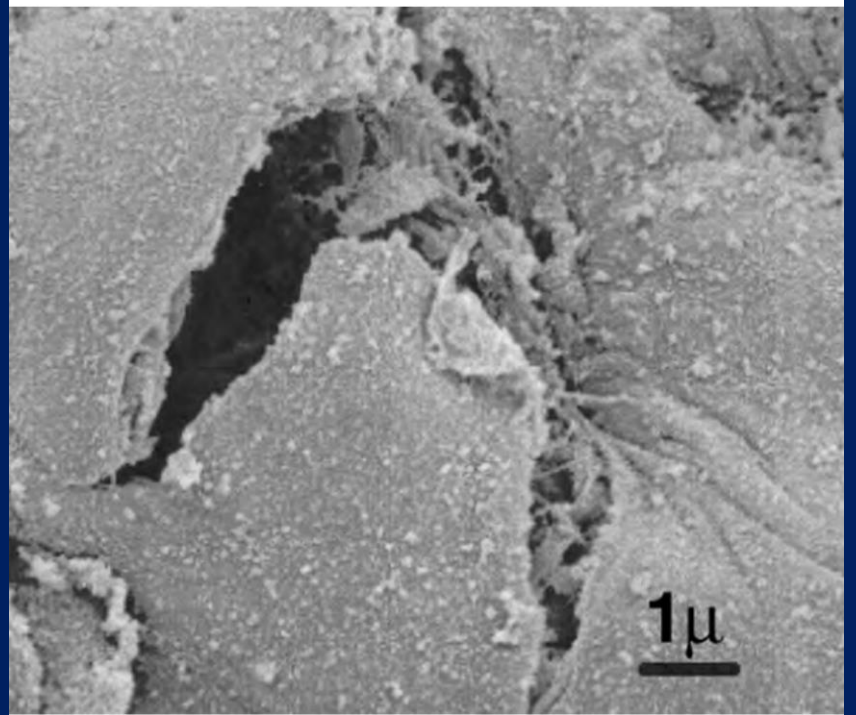
ventilator-induced "volutrauma"

pressure-volume curve of the lung



Post mortem specimen from a patient with ARDS





CXRay

- Alveolar and interstitial opacities
- Three-quarters of the lung fields

- ***Cardiogenic pulmonary edema***

ARDS

ARDS

Differential diagnosis of ARDS

- Cardiogenic pulmonary edema
- Diffuse pneumonia
- Alveolar hemorrhage
- Acute interstitial lung diseases
- Acute immunologic injury
- hypersensitivity pneumonitis
- Toxin injury
- radiation pneumonitis
- Neurogenic pulmonary edema

Management of ARDS

- Low tidal volumes (≤6 mL/kg)
- Positive endexpiratory pressure (PEEP)
- Adequate oxygenation with the lowest FIO2

RECOMMENDATIONS FOR ARDS THERAPIES

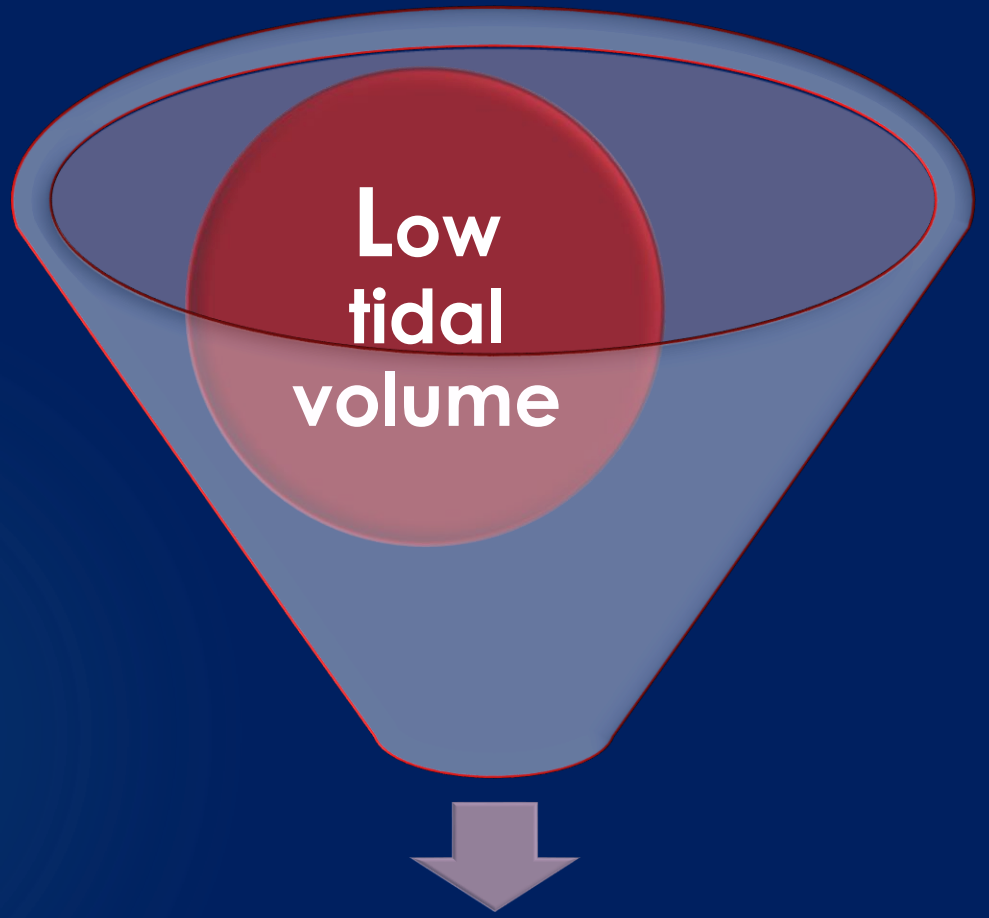
TABLE 322-3 EVIDENCE-BASED RECOMMENDATIONS FOR ARDS THERAPIES

Treatment	Recommendation[a]
Mechanical ventilation	
Low tidal volume	A
Minimized left atrial filling pressures	B
High-PEEP or "open lung"	C
Prone position	C
Recruitment maneuvers	C
High-frequency ventilation	D
ECMO	C
Early neuromuscular blockade	A
Glucocorticoid treatment	D
Surfactant replacement, inhaled NO, inhaled epoprostenol, and other anti-inflammatory therapy (e.g., ketoconazole, PGE1, NSAIDs)	D

TABLE 294-3 Evidence-Based Recommendations for ARDS Therapies

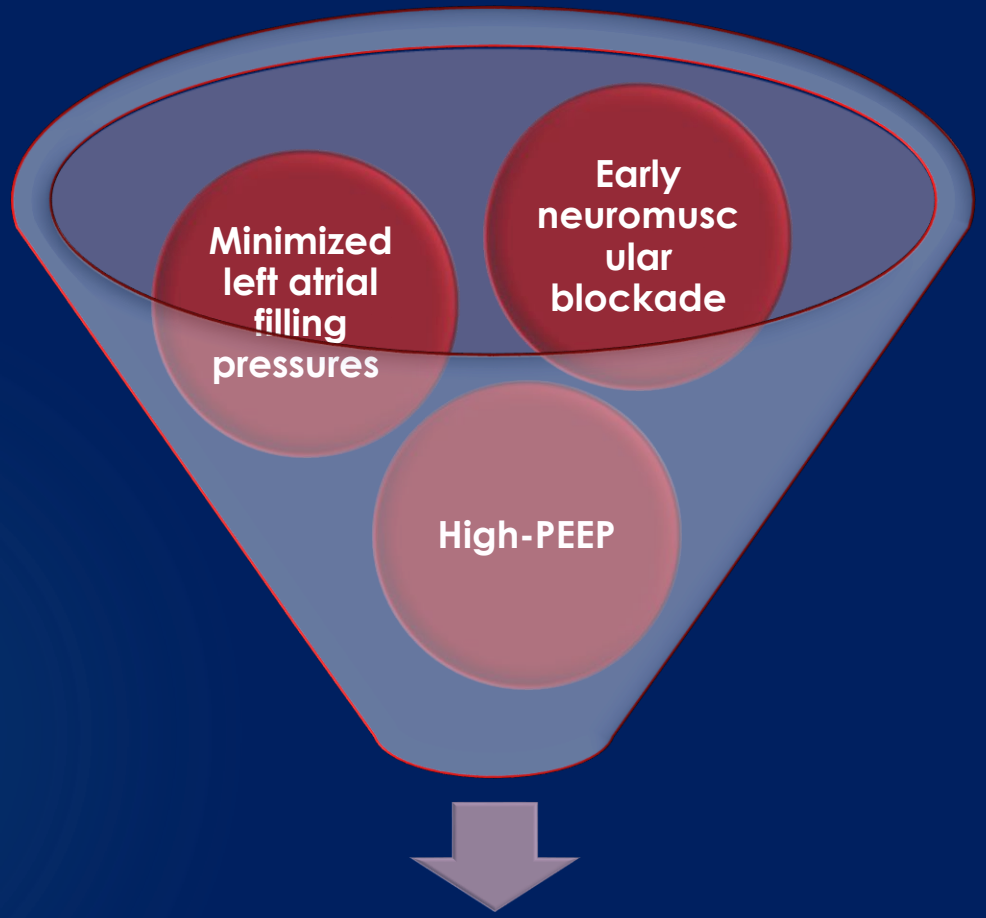
TREATMENT	RECOMMENDATION[a]
Mechanical ventilation	
Low tidal volume	A
Minimized left atrial filling pressures	B
High-PEEP or "open lung"	B[b]
Prone position	B[b]
Recruitment maneuvers	C
High-frequency ventilation	D
ECMO	B[b]
Early neuromuscular blockade	B[b]
Glucocorticoid treatment	D
Inhaled vasodilators (e.g., inhaled NO, inhaled epoprosteol)	C
Surfactant replacement, and other anti-inflammatory therapy (e.g., ketoconazole, PGE_1, NSAIDs)	D

Recommendation A



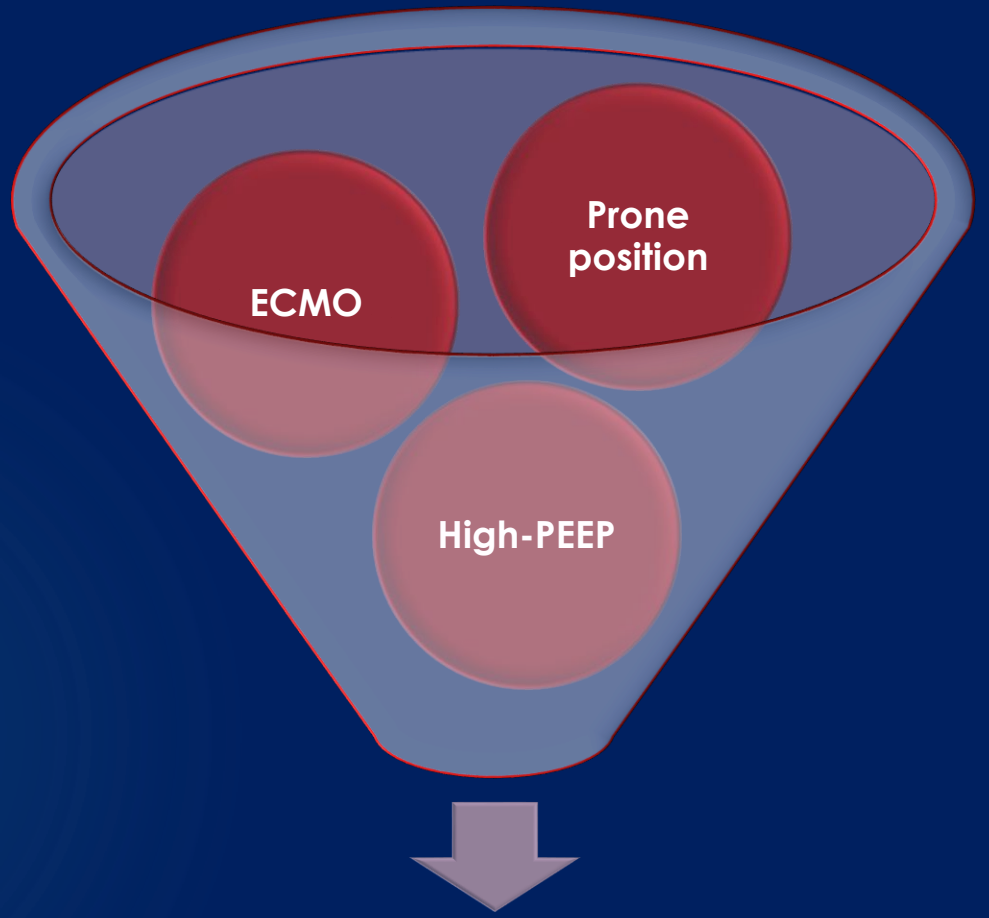
Recommended Therapy

Recommendation B



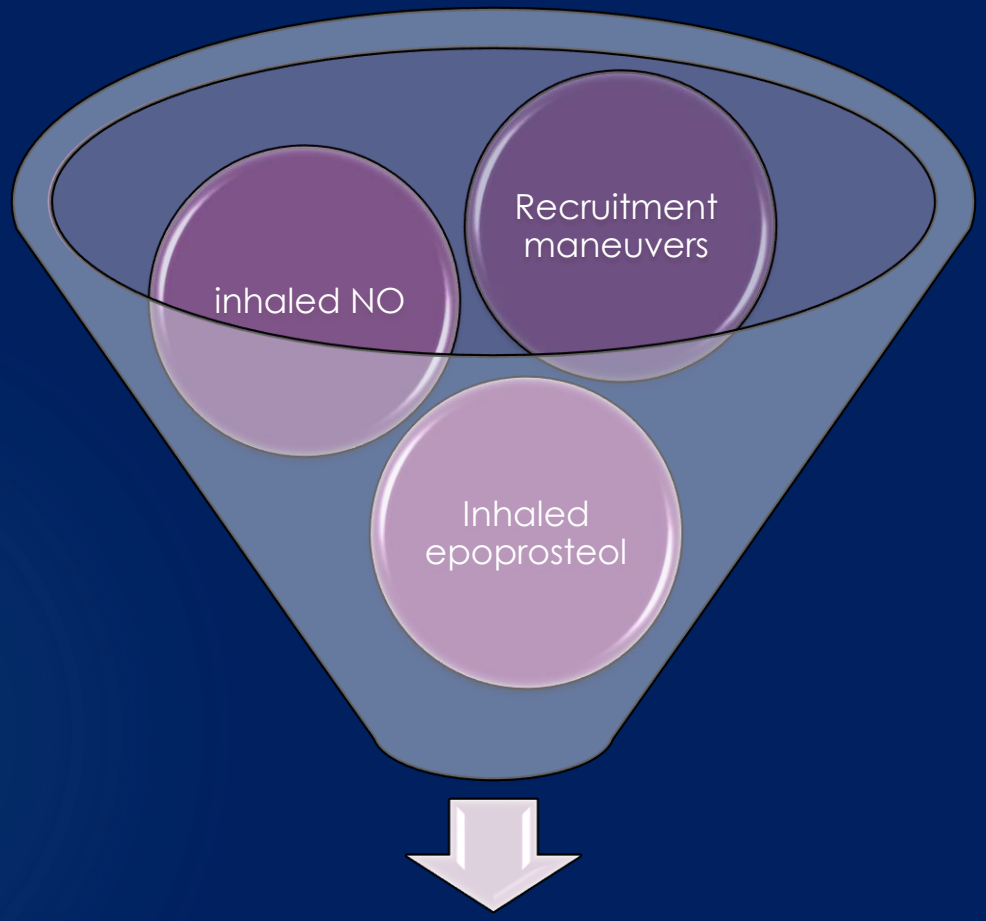
Recommended Therapy

Recommendation B



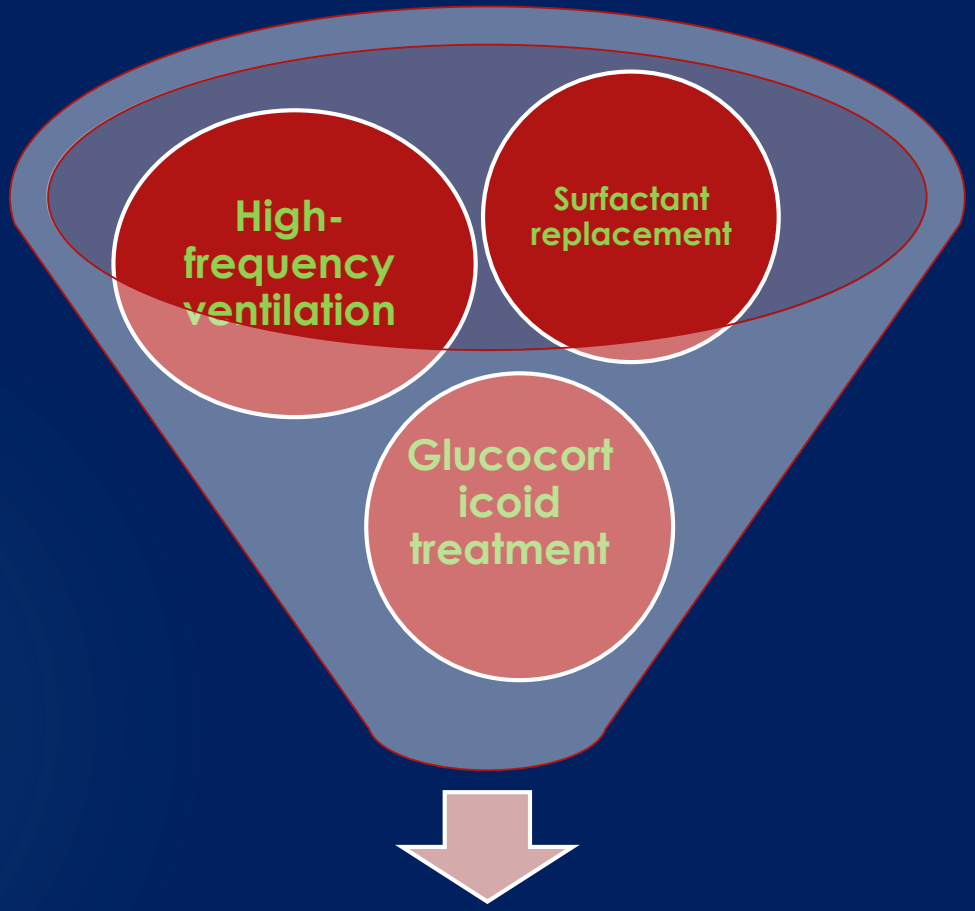
Recommended Therapy

Recommendation C



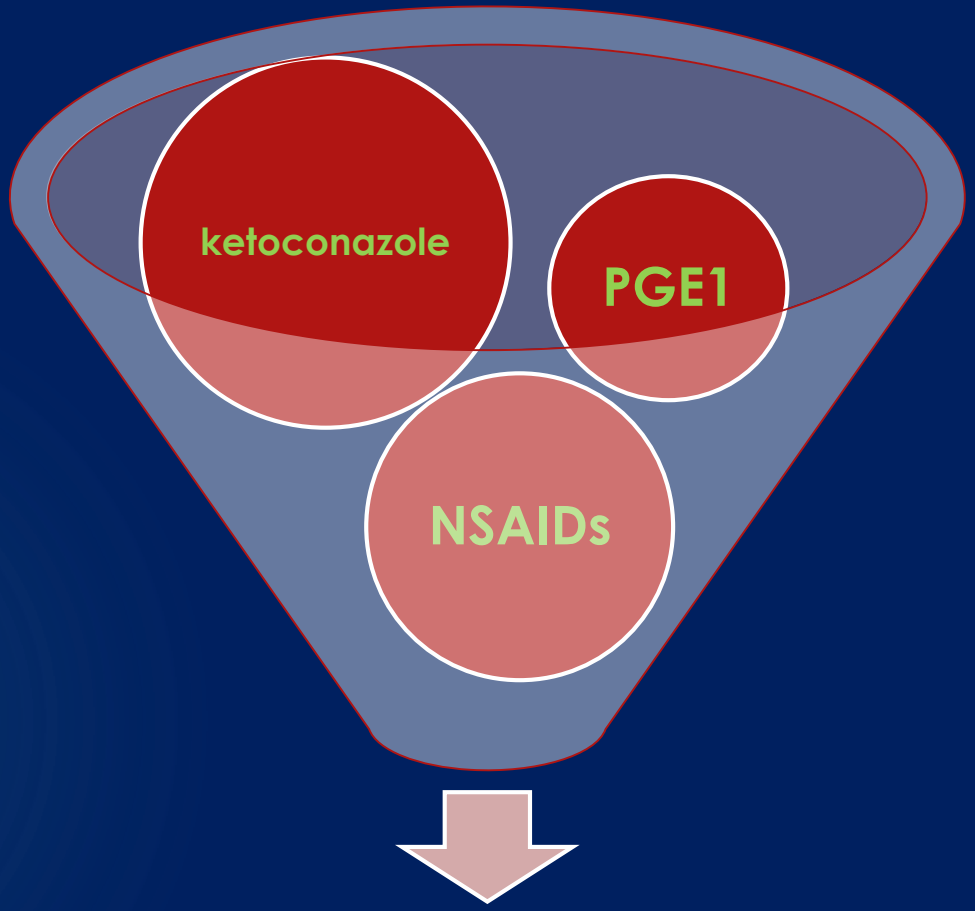
recommended only as alternative therapy

Recommendation D



Not Recommended

Recommendation D



Not Recommended

TREATMENT
GENERAL PRINCIPLES

- **care of critically ill patients**

1. recognition and treatment of underlying disorders
   - (e.g., pneumonia, sepsis, aspiration, trauma);
2. minimization of unnecessary procedures
3. "bundled care" approaches
   - Prophylaxis against VTE, GIB, aspiration, excessive sedation
   - prolonged MV, and CV catheter infections;
4. Nosocomial infections
5. Adequate nutrition

MANAGEMENT OF MECHANICAL VENTILATION

- Minimizing Ventilator-Induced Lung Injury
- Minimizing Atelectrauma by Prevention of Alveolar Collapse
- Prone Positioning
- Recruitment maneuvers
- Alternate modes of mechanical ventilation
- Lung-replacement therapy with ECMO

Minimizing Atelectrauma by Prevention of Alveolar Collapse

- alveolar and interstitial fluid
- Loss of surfactant

- reduction of lung compliance.

Minimizing Atelectrauma by Prevention of Alveolar Collapse

- significant alveolar collapse can occur at end-expiration
- Impairment of oxygenation
- positive end-expiratory pressure (PEEP)

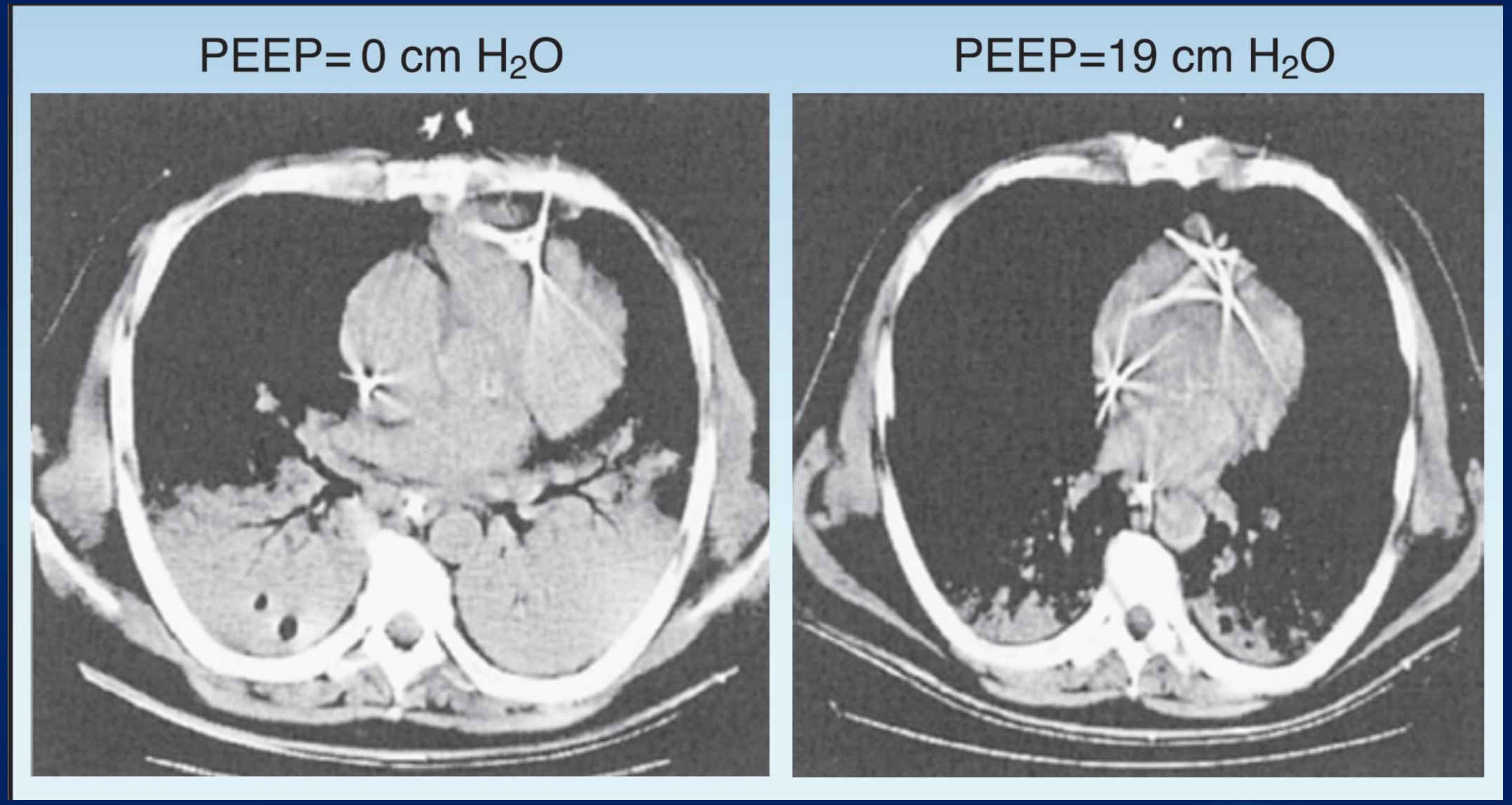
- minimize Fio2 (inspired O2 percentage)
- provide adequate Pao2
- without causing alveolar overdistention.

“best PEEP”

- lower inflection point
- esophageal pressures to estimate transpulmonary pressure

- Alveolar recruitment
- Minimizes alveolar overdistention
- hemodynamic instability
- adequate Pao2 while minimizing Fio2

“best PEEP”



Prone Positioning

- MV in the prone position
  - improved arterial oxygenation without a mortality benefit
  - significant reduction in 28-day mortality (32.8 to 16%)

- requires a critical-care team
  - accidental endotracheal extubation
  - loss of central venous catheters
  - orthopedic injury

FLUID MANAGEMENT ARDS

- Increased pulmonary vascular permeability
- impaired vascular integrity
- increasing left atrial pressure

aggressive attempts to

- reduce left atrial filling pressures with fluid restriction and diuretics
- important aspect of ARDS management
- hypotension and hypoperfusion of critical organs (kidneys)

NEUROMUSCULAR BLOCKADE In severe ARDS

- sedation alone
- early neuromuscular blockade (with cisatracurium besylate) for 48 h,

- increased survival and ventilator-free days
- without increasing ICU-acquired paresis

GLUCOCORTICOIDS

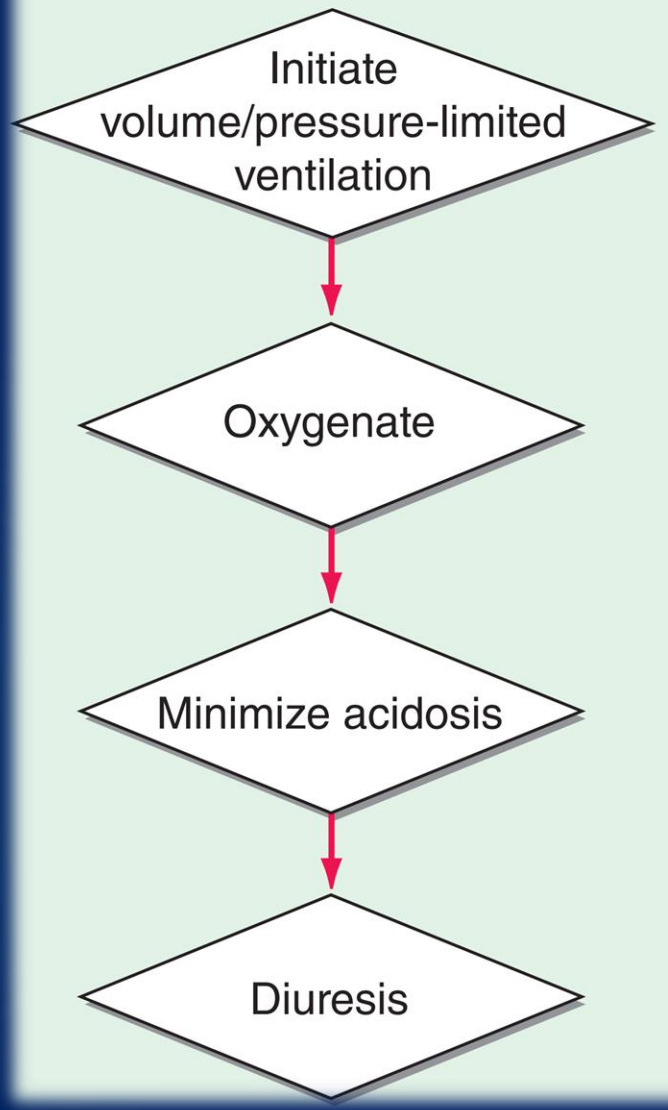
- Current evidence does not support the routine use

OTHER THERAPIES proved disappointing

- Surfactant replacement
- Pulmonary vasodilators
  - inhaled nitric oxide
  - inhaled epoprostenol sodium

- transiently improve oxygenation
- not been shown to improve survival or decrease time on MV

Initial Management of ARDS



Goals and Limits:

Tidal volume ≤ 6 ml/kg PBW
Plateau pressure ≤ 30 cmH_2O
RR ≤ 35 bpm

FIO_2 ≤ 0.6
SpO_2 88 – 95%

pH ≥ 7.30
RR ≤ 35 bpm

MAP ≥ 65 mmHg
Avoid hypoperfusion

PROGNOSIS

- hospital mortality estimates for ARDS
  - 34.9% for mild ARDS
  - 40.3% for moderate ARDS
  - 46.1% with severe ARDS
- mortality in ARDS is largely attributable to nonpulmonary causes
  - sepsis and nonpulmonary organ failure >80% of deaths

PROGNOSIS

- The major risk factors for ARDS mortality are nonpulmonary
- Advanced age > 75 years
  - mortality risk (~60%) than those < 45 (~20%)

- patients >60 years of age with ARDS and sepsis
  - threefold higher mortality risk than those < 60

PROGNOSIS
Other risk factors

- preexisting organ dysfunction
  - Chronic liver disease
  - Cirrhosis
  - Chronic alcohol abuse
  - Chronic immunosuppression
- Direct lung injury
  - Pneumonia
  - pulmonary contusion
  - Aspiration

twice as likely to die

PROGNOSIS
Other risk factors

- surgical and trauma patients with ARDS
- without direct lung injury

- higher survival rate than other ARDS patients

ARDS

- Increasing severity of ARDS

- predicts increased mortality

predicting ARDS mortality

1. Level of PEEP (≥10 cm H2O)
2. Respiratory system compliance (≤ 40 mL/cm H2O)
3. Extent of alveolar infiltrates on chest radiography
4. Corrected expired volume per minute (≥10 L/min)
   - (as a surrogate measure of dead space).

ARDS

- ***do not usually die of refractory hypoxemia***

- most patients with fatal ARDS die of
- ***sepsis and multiorgan failure***

Functional Recovery in ARDS Survivors

- prolonged respiratory failure
- mechanical ventilation for survival
- maximal lung function within 6 months
- One year after endotracheal extubation
  - >1/3 of ARDS survivors have normal PFT and DLCO
  - only mild abnormalities in pulmonary function
- Unlike mortality risk recovery of lung function
  - extent of lung injury in early ARDS

Less recovery of pulmonary function

- Low static respiratory compliance
- high levels of required PEEP
- longer durations of MV
- high lung injury scores

- ***Psychological problems***
- Depression
- Posttraumatic stress disorder

Therapy
Supportive Care

- treat the underlying cause
- antibiotics
- surgical débridement and drainage
- sepsis of unknown origin, both the lung and the abdomen
- prevention of complications
- nutrition, ventilation
- prophylaxis against gastrointestinal stress ulceration
- prophylaxis against deep venous thrombosis

Complications

- ***ventilator-associated pneumonia*** 30% to 65%
- > 5 to 7 days after the onset of MV
- nonfermenting gram-negative rods
- methicillin-resistant Staphylococcus aureus
- Enterobacteriaceae

- ***barotrauma*** 10% or less
- Pneumothorax
- Pneumomediastinum
- Subcutaneous emphysema

long-term complications in survivors

- Neuromuscular
- Psychosocial